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MANAGEMENT OF THE  
COCOANUT GROVE BURNS\*  
AT THE  
MASSACHUSETTS GENERAL HOSPITAL

*By the Following Members of the Staff*

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*And Their Staff Associates*



49 SUBJECTS IN COLOR ON 8 PLATES AND  
64 ILLUSTRATIONS IN BLACK AND WHITE

PHILADELPHIA

LONDON

MONTREAL

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SECOND IMPRESSION

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# Prologue\*

BY EDWARD D. CHURCHILL, COL. M. C. A. U. S.

*Consulting Surgeon North African Theatre of Operations*  
May 24 1945

REGARDING an injury as something apart from the body itself is a fallacy that has plagued the surgeon since his craft originated. The more external the lesion the more remote from the remainder of the body it appears and the more persistent becomes the effort to assist the natural processes of healing. Surgeons (and physicians) will apply chemical substances to the skin that they hesitate to place into a deep wound and will apply medicaments to the wound that they would not consider harmless in the deeper recesses of body cavities. Through years of hard experience it has been learned that the peritoneum has seemingly miraculous powers of resistance to infection if spared the added insult of chemical and mechanical trauma. The same is true of muscles and of the skin itself but the learning of this lesson has been delayed largely because violation is not attended by such immediate and catastrophic results as is the case with the peritoneum.

Again traumatic injuries lead the surgeon into a pitfall because they are so obviously of external origin. The cause is obvious and external so the remedy must be simple and local.

The local management of burns has for years revolved about these fallacious concepts. Fortunately the constitutional effects of severe burns have become clearly recognized although not clearly understood. The resulting reduction in deaths from loss of body fluids, anemia, malnutrition and infection has been an outstanding achievement of modern surgery.

As one explores the history of the local treatment of burns many remedies are found that are purely empirical. Others particularly those employed by reputable surgeons are supported by a perfectly logical rationalization in terms of the prevalent doctrine of the times. Turpentine is applied because the tissues have been thrown into a violent commotion by heat and this commotion must not be abruptly reversed but maintained at a high pitch for a longer time. Cerate, sweet oil, soothing balsams and later carron oil were emollients designed to reverse more quickly the agitated state of the tissues and presumably also the agitation of the patient himself. With the advent of Listerism carbolic acid is added to the bland and emollient oil in order to kill the germs. When patients were succumbing to the effects of traumatic toxemia tannic acid is introduced to precipitate these toxic

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\* To the Monograph on the Management of the Coconut Grove Burns at the Massachusetts General Hospital. Written in North Africa, it arrived too late to be included in the June issue of the *Annals of Surgery*.

## PROLOGUE

protein substances in situ. Later, when surgeons were paying more attention to body fluids, the virtue of tannic acid is found in the prevention of fluid loss from the weeping surface of the burn. When aniline dyes were taken from their rôle of staining dead tissues in the pathologic laboratory and used in the clinic on living tissues, gentian violet is found to produce an eschar similar to that formed by tannic acid but with the added virtue of killing Gram positive bacteria. Counterstains are added to take care of the Gram negative group. Now that the sulfonamides are in full voice, they are either dusted on in pure form or incorporated in ointments. And so it goes.

Since being in North Africa I have seen a great many burns—mostly accidental, as this is a petrol war. I have also seen almost as many different methods of local treatment as there are burns. In fact, a single burn may recapitulate many pages of the history of the treatment of burns, as the patient is moved about the Theatre of Operations. I have even seen burns treated by the native population with fresh cow dung—and they healed very kindly at that. Perhaps "there is something in it,"—certainly some vitamin or other biotic can be isolated from cow manure by painstaking research given a little time and the necessary grant-in-aid.

In the Army, the Battalion Surgeon is the important fellow. He is right down at the surgical grass roots of the war. He must get results and get them quickly, and what is more, he must work with what can be carried on his shoulder in a kit bag. Here are comments regarding the treatment of burns that can be kept in the combat area by two Battalion Surgeons.

Number One "We believe in covering them up quickly with a big dressing and then pouring cold water on the dressing. It is not always sterile water either, usually out of my canteen. Do I clean them off? No, I never scrub anything up there at the front." (See Chisolm's excellent *Military Surgery of the Confederate Army* for further description of the cold water treatment of wounds. There is something in it.)

Number Two "You know I tried all of these various treatments for burns, but the one I think works best is just boric ointment. It takes the pain right away, and they seem to do better. I don't suppose I should be using it, they say you shouldn't put grease on burns. But up there at the front you begin to wonder about a good many things you were taught in Medical School."

Events move rapidly in war, and I am glad to report a heartening trend toward sanity in an otherwise schizophrenic world. When external violence reaches epidemic proportions one is forced to think in practical and simple terms. So it has happened with burns.

Although the Cocoanut Grove Disaster took place in Boston the method used for the treatment of burns was inspired by the war already in progress some months. The nightclub patrons were cared for by a method that might be applied to soldiers and sailors of an assault force on a sea beach. It is true that frills were added and elaborate laboratory tests carried out to prove to ourselves and others that the patients were not suffering ill consequences. One worries more about simplifying treatment than elaborating it.

## PROLOGUE

The essentials of the treatment are being applied daily in North Africa and it has been comforting to be able to assure the Battalion Surgeon that it is not something he shouldn't be doing.

It is difficult to write a prologue to a collection of papers that I have never seen even in preliminary draft. During the catastrophe and for some weeks afterward we were concerned only with observing and recording facts. I hope my colleagues have been cautious in formulating conclusions. Even if modest conclusions are likely to be wrong or subject to modification before the ink is dry.

Opinions and impressions are plentiful and cheap particularly under the emotional tension of a world at war. Facts are rare and precious. Complete data are next to impossible to obtain under combat conditions. Observations are fragmentary at the best. Yet the surgeons of the United States Army are already facing a barrage of surgical opinions and impressions from our many Theatres of Operations.

It is essential to preserve an open mind for what is true under one set of conditions will not hold for a different set of conditions. What may be successful in the Pacific may not be at all applicable in other hands in North Africa or in England. American surgeons are at work in India and Iceland, Brazil and the British Isles, Alaska, Australia and Africa. They are in both hemispheres down under the Equator and within the Arctic Circle. Only when we have all returned each with a small piece of the puzzle will it be possible to start to fit the pieces together and see the picture as a whole. In the meantime we must be content to observe and record so that our craft may rescue something constructive from a catastrophe that in other aspects represents the acme of destruction.

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# Contents

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OLIVER COPE M.D.

Foreword

NATHANIEL W. LAXON M.D.

The Problems of the Hospital Administration	3
<i>The Cocoanut Grove Disaster</i>	3
<i>A Brief Account of the Services of the Massachusetts General Hospital in Connection with the Cocoanut Grove Disaster</i>	3
<i>We Learn from Our Mistakes as Well as from Our Successes—What Has the Massachusetts General Hospital Learned from the Cocoanut Grove Disaster?</i>	6

IDA M. CANNON I.H.D.

Social Service Activities	17
---------------------------	----

STANLEY COBB M.D. AND ERICH LINDEMANN M.D.

Neuropsychiatric Observations	14
<i>Psychotic Conditions Primarily Psychogenic</i>	16
<i>Psychoneuroses</i>	17
<i>Reactions to Bereavement</i>	18
<i>Special Studies</i>	19
<i>Suggestions for Future Emergencies</i>	23
<i>References</i>	24

HENRY K. BEECHER M.D.

Resuscitation and Sedation of Patients with Burns Which Include the Airway	25
<i>Common Causes of Hyperactivity</i>	25
<i>Therapy of the Causes of Hyperactivity</i>	26
<i>Pain</i>	27
<i>Fear and Hysteria</i>	28
<i>Anoxia from an Inadequate Airway</i>	28
<i>Anoxia from Inadequate Transport of Oxygen by the Blood</i>	30



## CONTENTS

<i>Observation of Patients Following Immediate Therapy</i>	31
<i>Delayed Reactions</i>	31
<i>Need for Future Work</i>	32
<i>Summary and Conclusions</i>	32
<i>References</i>	33

JOSEPH C AUB, M D , HELEN PITTMAN, M D , AND AUSTIN M BRUES, M D

<i>The Pulmonary Complications A Clinical Description</i>	34
<i>Conclusions</i>	40

RICHARD SCHATZKI, M D

<i>Roentgenologic Report of the Pulmonary Lesions</i>	41
<i>Patients with Pulmonary Pathology by Roentgenogram</i>	43
<i>Analysis of the Roentgenologic Findings</i>	50
<i>Patients Showing No Roentgenologic Pulmonary Changes</i>	63
<i>Discussion</i>	63
<i>Summary</i>	63
<i>References</i>	64

TRACY B MALLORY, M D , AND WILLIAM J BRICKLEY, M D

<i>Pathology With Special Reference to the Pulmonary Lesions</i>	65
<i>Cases Dead on Arrival</i>	65
<i>Cases Treated in the Hospital</i>	68
<i>Summary of Postmortem Findings</i>	80
<i>Discussion</i>	84
<i>References</i>	84

OLIVER COPE, M D

<i>The Treatment of the Surface Burns</i>	85
<i>The Surface Treatment Used on Coconut Grove Patients</i>	85
<i>Conclusions</i>	93
<i>References</i>	93

CHAMP LYONS, M D

<i>Problems of Infection and Chemotherapy</i>	94
<i>Factual Data</i>	94
<i>Inference from Clinical Observations</i>	100
<i>Conclusions</i>	102
<i>References</i>	102

# CONTENTS

BRADFORD CANNON M D

Procedures in Rehabilitation of the Severely Burned	103
<i>Operative Procedures</i>	105
<i>Progress and Results</i>	108
<i>Comment</i>	109

ARTHUR I WATKINS M D

A Note on Physical Therapy	111
<i>Results</i>	112
<i>Comment</i>	112

OLIVER COFF M D AND FREDERIC W RHINELANDER M D

The Problem of Burn Shock Complicated by Pulmonary Damage	115
<i>Conclusions</i>	127
<i>References</i>	128

LAMAR SOUTHER M D

A Note on the Blood Bank	128
--------------------------	-----

FRANCIS D MOORE M D

A Note on the Thrombophlebitis Encountered	131
<i>Discussion</i>	136
<i>Conclusions</i>	136

OLIVER COPE M D IRA T NATHANSON M D

G MARGARET ROURKE A B AND HILDEGARD WILSON PH D

Metabolic Observations	137
<i>Experiments</i>	142
<i>Clinical Correlations</i>	149
<i>Comment</i>	149
<i>Summary and Conclusions</i>	155
<i>References</i>	158
Protocols	159
Index	167



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# *Management of the Cocoanut Grove Burns at the Massachusetts General Hospital*

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## FOREWORD

On Saturday evening November 28 1942 almost a year after America entered the war a disastrous fire occurred in the Cocoanut Grove a Boston night club. As a result 491 people lost their lives and many were injured. Of the casualties dead and living 114 were brought to the Massachusetts General Hospital within a period of two hours. Had such a catastrophe taken place before Pearl Harbor the hospital would have been swamped. As it was the injured found the staff prepared for the war had made us catastrophe minded. The hospital was well prepared partly as a result of the foresight of Dr. N. W. Laxon Director and of Dr. E. D. Churchill Chief of the West Surgical Service who had given careful thought to the problems of the hospital faced with a disaster and partly to the good fortune that an active research program in burns was in progress at the hospital.

Early in 1942 two research projects were undertaken at this hospital under contract with the Office of Scientific Research and Development of the United States Government. One dealt with infections in compound wounds and burns and the other with the physiology of burns. In both of these projects a number of the staff had taken part. All patients with burns whether on the public or private wards were used in the study. Modes of therapy were tried out and discussed at repeated staff conferences. A plan of therapy for burns suited to use in a catastrophe was developed and decided upon. When the victims of the Cocoanut Grove fire arrived the treatment was ready and it was applied to all.

As in many disasters the type of injury encountered in the casualties of the Cocoanut Grove fire conformed to a pattern. The lungs and airways were severely damaged perhaps both by heat and irritating gases. The external burns were for the most part limited to skin surfaces not covered by clothing. Even the burns of eyes were of a pattern the lower half of the

## FOREWORD

cornea of both eyes In spite of the crowding and panic, there were no fractures.

The injury of the lungs presented the most pressing problem of therapy The complications encountered were similar to those resulting from inhalation of certain war gases They were also like those described following the disastrous fire at the Cleveland Clinic in 1929, believed due to toxic nitrogen gases released from burning roentgen films This Cleveland disaster has come to be considered unique, for as a result of it the chemical content of roentgen film had been changed and all old film destroyed throughout the country From the experience of the Cocoanut Grove fire, we know that such pulmonary complications are to be found not solely in warfare, but may be encountered at any moment in civilian life The experience with these unusual lung complications is worthy of recording Valuable material has been gathered dealing with the nature of the pathologic processes The Massachusetts General Hospital and Harvard University have given unstintingly to insure a reaping of as much useful knowledge as possible

The treatment used on the burns of the skin was unorthodox but the results were gratifying Its simplicity has much to recommend it when large numbers of burns are encountered in a disaster Because the method should prove of interest to the Armed Forces and to those charged with the responsibility of caring for civilian catastrophes, it deserves description particularly at this point of the World War

The monograph has been arranged as a series of papers by the members of the staff who were responsible in their particular fields The first three articles deal with the problems of administration and psychiatry peculiar to a civilian disaster The next four recount the course and treatment of the lung injuries The last five deal with the various aspects of the surface burns

A brief protocol of each case, numbered consecutively, follows the last article The same case numbers are used throughout all articles Where cases are referred to and no details are given in the article, they are to be found in the protocol

The authors dedicate this monograph to all those who labored with them yet remain anonymous, and to Dr Churchill who guided us before he was called to serve in the Army

OLIVER COPE, M D

The Massachusetts General Hospital acknowledges with gratitude the aid of the Josiah Macy, Jr Foundation in sharing the cost of publication of the colored photographs

# THE PROBLEMS OF THE HOSPITAL ADMINISTRATION

NATHANIEL W. FAXON, M.D.

## I—THE COCOANUT GROVE DISASTER

THE COCOANUT GROVE was a typical night club. It was a one story stucco and brick building with low ceilings and inflammable hangings and decorations. On the evening of November 28, 1942, it was filled with an unusually large Saturday night crowd. The reported capacity was 600; the estimated number present was 1000. The several bars were crowded; the tables were filled to capacity; with every available bit of floor space occupied. The floor show was about to begin.

Fire started in the Melody Lounge, a basement cocktail lounge, about 10:15 P.M. Feeding on combustible decorations, artificial coconut palms and cloth-covered ceilings and walls, it spread with great rapidity to the stairs, cutting off the only visible means of exit. Then it flashed across the ceiling of the main floor. People rushed for the main doorway, the only exit that they knew of, where a revolving door quickly jammed and some 200 victims were piled behind it. The flames then spread to the Broadway Cocktail Lounge where 100 more victims were trapped behind a door swinging the wrong way, which blocked access to the outside doorway. A door leading from the main floor was partially opened by an employee through which a few escaped. Other exits were hidden by hangings and also locked. All agree that the spread of flame was rapid, with much smoke and noxious gases and the lights went out quickly.

The fire department responded promptly and in adequate force. They opened doors, broke in windows, extinguished the fire and rescued as promptly as possible all who were still alive, but 491 people lost their lives, either there or later in hospitals. Many others were seriously injured. One hundred and eighty-one living victims were taken to hospitals together with nearly 300 who were dead on arrival. Thirty-nine living patients arrived at the Massachusetts General Hospital, 131 at the Boston City Hospital and the 11 others at various nearby hospitals. During the next two weeks, 39 patients died in hospitals—seven at the Massachusetts General Hospital and 32 at the Boston City Hospital.

## II—A BRIEF ACCOUNT OF THE SERVICES OF THE MASSACHUSETTS GENERAL HOSPITAL IN CONNECTION WITH THE COCOANUT GROVE DISASTER

The Coconut Grove fire started about 10:15 P.M. Saturday, November 28, 1942. The first patients arrived at the Emergency Ward of the Hospital at 10:30 P.M. Shortly thereafter the hospital was notified of the disaster and asked to be ready for a large number of patients. The hospital organization set up under Civilian Defense for the handling of war casualties was immediately put into operation. The House Staff and nurses on duty were

called to the Emergency Ward, Teams for Burns and Resuscitation were summoned, members of the visiting staff, nurses off duty, social workers volunteers, orderlies, and others were notified By 11.15 P M nearly the entire organization had been assembled, and volunteers continued to arrive during the night

The Emergency Ward was immediately cleared of all other patients, but it was soon realized that its facilities would be overtaxed, and the sixth floor of the White Building, containing 40 beds, was evacuated Thirty surgical patients were removed to other beds in the hospital

Between 10 30 P M and 12 45 A M , 114 casualties were received Of these, 75 were either dead on arrival, or died within the first few minutes of uncontrollable anoxia

The 39 living patients showed, besides burns of varying degrees, the effects of cold, exposure, fright, shock, and partial asphyxia Clothing was dripping wet, exposed surfaces grimy and blackened Some were quiet and cooperative A few were comatose, others were greatly agitated, requiring restraint In some this was due to hysteria, in others to cerebral anoxia (lack of oxygen supply to the brain) There was little or no evidence of intoxication There were no fractures and only slight trauma of soft parts

Wet clothing was immediately removed, burned surfaces covered with sterile towels and the patients wrapped in blankets Morphine was given subcutaneously to all

Meanwhile, four members of the House Staff had been stationed at the entrance of the hospital to determine whether those admitted were living or dead Those pronounced dead were carried directly to an emergency morgue established in the large Brick Corridor, the bodies covered with sheets and the area screened off

Some of the dead showed no burns, others showed burns of varying degrees, but death evidently had come from asphyxia in most cases Many showed the cherry-red color indicative of carbon monoxide asphyxia A few were severely burned, one almost beyond recognition Identification of the dead was started at once, and all but two of the men were identified by 5 00 A M Identification of the women was very difficult on account of the lack of identifying data in their clothing They were identified only by direct inspection by relatives or friends

Preliminary treatment having been given to the living in the Emergency Ward, they were moved to White-6 By 1.30 A M all the living, 39 in number, had been put to bed in this ward Burns had been dressed, shock was being treated, and asphyxia cases given oxygen therapy The Emergency Ward was cleared and ready for more admissions. Thirty of the 39 patients had surface burns of clinical significance and many showed evidence of damage to the respiratory tract In some this was very severe In some cases it developed 24 hours later In one case artificial respiration was required for the first six hours The patient recovered Five cases required tracheotomy and in one or two tracheal intubation was performed

The surface burns were treated by a single method. There was no cleansing or débridement. The burned surfaces were covered with gauze impregnated with boric petrolatum and a voluminous dressing applied with elastic pressure bandages. Splints of folded newspaper were used for fore arms and hands. Eyes were examined by Staff and Residents from the Massachusetts Eye and Ear Infirmary and appropriate treatment instituted in the cases with lesions involving the cornea.

Blood plasma from the hospital blood bank was administered to 29 patients. By 1:00 A.M. all patients in impending shock were receiving plasma. Salt solution and glucose solution had been previously administered intravenously and was continued thereafter for long periods in many cases. In the first 24 hours 120 units of frozen plasma were used; in three days a total of 147 units. In addition 16 whole blood transfusions were given in the first week where there was reduced oxygen capacity. Teams of interns and nurses under the direction of surgeons, residents and the chief anesthesiologist were assigned to individual patients and to groups and were in constant attendance. There were 20 trained nurses on eight hour duty on this ward or 60 each 24 hours. Oxygen therapy, directly by catheter or in tents, was given in appropriate cases; carbon dioxide five to seven per cent in oxygen was given to three patients showing evidence of carbon monoxide poisoning. Sterile suction tubes for intratracheal intubation were used in several cases. In addition to the more intricate treatment routine intravenous medication, blood pressure determinations, blood tests, etc. were carried out.

Sulfadiazine had been given intravenously by 2:00 A.M. Sunday to all patients including those without burns and its administration was continued thereafter in appropriate amounts. All patients having been previously serum tested were given antitetanic serum except Army and Navy personnel and those with serious pulmonary lesions.

By 3:00 A.M. Sunday a list of all the living, with names and addresses had been made and given to police and press.

During the first three days seven patients died. These were all cases showing severe respiratory tract damage from inhalation of flame or fumes. No cases of true bacterial pneumonia developed.

All the patients were segregated and isolated on the sixth floor of the White Building. Admission to the ward was strictly limited with a doorkeeper in constant attendance. Besides professional staff and attendants only immediate relatives, clergy, family doctors and officials on errands of importance were admitted and all were masked and gowned.

The East solarium was converted into a dressing station where all dressings were done under the full aseptic precautions used in an operating room. Roentgenologic examinations of the chest were carried on in the South solarium on the same floor; all patients were so examined by 10:30 A.M. on Sunday and thereafter as indicated.

The clinical laboratories were manned immediately on the night of the disaster and were kept busy continually thereafter. Hematocrit and serum protein determinations to guide the administration of fluids and plasma were



## NATHANIEL W FAXON

available that night, five times in all in the first 24 hours. Determinations of oxygen and carbon dioxide content of blood, oxygen capacity, nonprotein nitrogen, prothrombin time, blood chloride, phosphorus, sodium, and other tests, including various bacteriologic examinations, were also made.

The pathologic laboratory was called into action in making tissue examinations and later in performing autopsies.

Invaluable service was rendered by members of the Social Service Department. They helped in various places where needed. Their training and skill in dealing with people emotionally disturbed fitted them for the tasks of interviewing distracted relatives and friends of patients, answering innumerable telephone calls. They also helped in the identification of the living and the dead.

The services of volunteers from the Ladies' Visiting Committee and the War Service Committee proved of great value. They were personable, poised and they knew the hospital.

Medical students assigned to the hospital, together with a group of Harvard undergraduates who had been doing volunteer work as orderly attendants for six months, some of whom were on duty, and others who came in afterward, rendered valiant aid that night and thereafter. They also knew the hospital.

The Red Cross functioned smoothly in the emergency. The motor ambulance brought casualties to the hospital. The Nurses' Aides were of immediate assistance that night, and the next day large numbers replaced and reinforced regular nurses on duty in other parts of the hospital. The Red Cross Canteen, in connection with the hospital dietary department, served coffee and sandwiches during that night, which was a great help in sustaining energy and morale of workers and giving comfort to relatives and friends.

Many private nurses from Baker Memorial and Phillips House contributed their services during spare time.

The Massachusetts Women's Defense Corps sent volunteers who aided in identification and also were of great help with the Blood Bank.

Over 100 outside nurses from neighboring institutions volunteered and were put to work.

The regular staff of the hospital, administrative, medical, surgical, special, nurses, orderlies, technicians, telephone operators, secretaries, janitors, porters, maids, and maintenance personnel labored long hours at night and Sunday without stint.

In conclusion, it may be said that the hospital organization met the emergency adequately and well. Much credit should be accorded to the Civilian Defense organization for having made the hospital "catastrophe-minded" beforehand. There was no shortage of supplies or equipment of any kind.

### III—WE LEARN FROM OUR MISTAKES AS WELL AS FROM OUR SUCCESS WHAT HAS THE MASSACHUSETTS GENERAL HOSPITAL LEARNED FROM THE COCONUT GROVE DISASTER?

I First of all, we have learned the value of anticipation and preparation.

Thanks to the efforts of the Massachusetts Committee for Public Safety or Civilian Defense we had been made 'catastrophe minded'. Our Staff and personnel had been organized as teams their duties carefully specified information regarding disaster management spread widely and practice mobilizations carried out. Although during the first two hours everything seemed to be in confusion because of the numbers of people hurrying about it was clear to those responsible that everyone was acting rapidly efficiently and intelligently. They knew what they should do and were doing it.

Furthermore we learned the value of having on hand what might have been considered an unnecessarily large quantity of supplies. Fortunately no shortage of anything was experienced.

2 The value of a well planned telephone service to notify administration staff nurses technicians maintenance and department heads should be especially emphasized. Too much thought and planning cannot be given to this service.

3 The necessity of the immediate examination and separation of the living and dead at the very entrance of the hospital. This was only realized after a number of dead had been sent to the Emergency Ward. At once four Medical house officers in teams of two were stationed at the Emergency Ward entrance for this purpose. It is important that two men should collaborate.

4 The organizing of teams of nurses and assistants for the undressing of patients of the affixing of identifying tags and for the care and marking of their clothing and belongings. Those entrusted with the medical or surgical treatment of patients have no time for this and the identification of patients separated from their clothing may be difficult. Likewise, clothing and valuables may get hopelessly mixed.

5 Special medical teams for the administration of morphine the treatment of shock by plasma and oxygen therapy were found to be valuable.

6 Prompt examination of the dead by competent pathologists. Medical Examiner or hospital pathologists may provide clinicians with valuable data which will aid in treatment of the living. Autopsies upon those dying after clinical appraisal of symptoms are particularly helpful. The findings provided by autopsies performed and authorized by the Medical Examiners of Boston were of great assistance.

7 Handling of the dead. From our experience we learned that an Emergency Morgue should be selected in advance, paying attention to accessibility isolation windows for ventilation and cooling. It should be at once placed in charge of some responsible person and a police guard obtained as soon as possible. Only persons bearing passes acceptable to the person in charge and to the police should thereafter be admitted. The bodies should be arranged in orderly fashion heads in the same direction sexes separated if possible and covered with sheets. Tags numbered consecutively (M G H 1 2 3 etc) bearing date and hour of arrival should be attached to the right wrist. The Medical Examiner or Coroner should

## NATHANIEL W FAXON

be consulted as soon as possible and his directions followed. If he so directs, identification of the dead may be carried out. The name of the dead person can then be added to the tag. Also, at that time, valuables, pocketbook and cards may be placed in an envelope bearing the same number as the tag and attached to the left wrist. No jewelry should be removed.

However, if the Medical Examiner so directs, a complete list of all valuables can be made by two persons, one preferably a police officer, the valuables placed in an envelope, signed by the two persons and placed in a safe.

8 A list of living casualties and a list of identified dead should be prepared as quickly as possible and sent to the Information Desk. There it should be arranged alphabetically, several typed sheets prepared, and the information given to the police and press. All inquiries should be directed to this Central Information Desk.

9 In a disaster of this type, where the injuries were all of the same kind, the importance of concentration of casualties in one group in one ward or floor where they can be under concentrated medical treatment and where isolation procedures may be set up if needed, was clearly demonstrated.

In this disaster, a problem was presented by the abrupt and unexpected confinement in the hospital of 39 seriously injured people of private patient status. Isolation precautions were considered imperative and this had to be explained to anxious families. Many requests were received for transfer to private rooms for private medical care at the hospital or other institutions. The policy was immediately announced that visits from family doctors or consultants at the request of patient or family would be welcomed. The local medical profession cooperated whole-heartedly. Doctors visited their patients, reassured them and advised them under no condition to consider removal. Medical information which had a bearing on their present condition was often given to the Staff.

It is the mature judgment of those who cared for these patients that this concentration and this isolation prevented, to a large degree, respiratory complications and permitted better treatment. Doctors, nurses, equipment and supplies were concentrated here, quickly available for emergency treatment. Time and labor were saved.

10 It is desirable to obtain speedily police assistance to control yard traffic and to guard hospital corridors and morgue.

11 The serving of coffee and sandwiches by the Dietary Department and the Red Cross Canteen was valuable in sustaining energy and morale of workers and comforting waiting relatives and friends.

12 Finally, we have learned the value of constantly maintaining, for use in peace or war, a hospital organization for the handling of emergency disaster, also the collection of an ample quantity of emergency supplies.

“An emergency anticipated and prepared for ceases to be an emergency.”

## SOCIAL SERVICE ACTIVITIES

IDA M CANNON I H D

FROM THE SOCIAL SERVICE DEPARTMENT OF THE MASSACHUSETTS GENERAL HOSPITAL, BOSTON

THE CASUALTY PREPAREDNESS PROGRAM at the Massachusetts General Hospital delegated to the Social Service Department several areas of responsibility in keeping with their recognized function. These were

- I Identification and registration of victims in assistance to Hospital Admitting Officers. Reporting list of victims to Master File conducted by the Committee on Public Safety.
- II Arranging for transfer of such patients as were suitable for discharge to their own homes, convalescent or nursing homes or other hospitals to relieve the wards for admission of victims.
- III Maintenance of Information and Advice Service to families and friends of victims. This to relieve the Hospital Information Service.
- IV Liaison with Red Cross Disaster Relief in their service to victims and with Public Safety District Information and Advice Bureaus.

Members of the Department had shared in practice with other hospital personnel in accordance with the Manual of the Casualty Preparedness program for war-caused disaster. Although the "pattern" of the Cocoanut Grove disaster differed from the type anticipated in practice the essential characteristics of social service activities were realized.

Social Service staff was not called promptly but by 12:30 A.M. Sunday four members of the Social Service staff were at the hospital and during Sunday 20 workers were involved. Twenty-four hour service was maintained for the first two days and 12 hour service for the first week.

Prompt shifting of patients from the especially chosen ward to other parts of the hospital had obviated the necessity for discharge of patients to make room for a flood of admissions. The urgency of admission of victims to the prepared special ward and the need for immediate treatment procedures made the usual process of identification *en route* impossible and necessitated completion of identification after patients reached the ward. A social worker assisted in listing the victims and arranged an alphabetical list for use of the Admitting Office and for report to the Master File. As there were two unconscious unidentified women on the ward she attended one of the doctors as one of the patients was aroused sufficiently to give her name. The other was identified by a patient nearby. By 3 A.M. all 39 living victims were correctly identified by name and address.

The first medical social worker arriving at the hospital a little before midnight was immediately pressed into service by the nurse at the Hospital Information Desk. Two and at times three social workers assisted here throughout the night and morning. Three telephones brought incessant

## NATHANIEL W FAXON

be consulted as soon as possible and his directions followed. If he so directs, identification of the dead may be carried out. The name of the dead person can then be added to the tag. Also, at that time, valuables, pocketbook and cards may be placed in an envelope bearing the same number as the tag and attached to the left wrist. No jewelry should be removed.

However, if the Medical Examiner so directs, a complete list of all valuables can be made by two persons, one preferably a police officer, the valuables placed in an envelope, signed by the two persons and placed in a safe.

8 A list of living casualties and a list of identified dead should be prepared as quickly as possible and sent to the Information Desk. There it should be arranged alphabetically, several typed sheets prepared, and the information given to the police and press. All inquiries should be directed to this Central Information Desk.

9 In a disaster of this type, where the injuries were all of the same kind, the importance of concentration of casualties in one group in one ward or floor where they can be under concentrated medical treatment and where isolation procedures may be set up if needed, was clearly demonstrated.

In this disaster, a problem was presented by the abrupt and unexpected confinement in the hospital of 39 seriously injured people of private patient status. Isolation precautions were considered imperative and this had to be explained to anxious families. Many requests were received for transfer to private rooms for private medical care at the hospital or other institutions. The policy was immediately announced that visits from family doctors or consultants at the request of patient or family would be welcomed. The local medical profession cooperated whole-heartedly. Doctors visited their patients, reassured them and advised them under no condition to consider removal. Medical information which had a bearing on their present condition was often given to the Staff.

It is the mature judgment of those who cared for these patients that this concentration and this isolation prevented, to a large degree, respiratory complications and permitted better treatment. Doctors, nurses, equipment and supplies were concentrated here, quickly available for emergency treatment. Time and labor were saved.

10 It is desirable to obtain speedily police assistance to control yard traffic and to guard hospital corridors and morgue.

11 The serving of coffee and sandwiches by the Dietary Department and the Red Cross Canteen was valuable in sustaining energy and morale of workers and comforting waiting relatives and friends.

12 Finally, we have learned the value of constantly maintaining, for use in peace or war, a hospital organization for the handling of emergency disaster, also the collection of an ample quantity of emergency supplies.

"An emergency anticipated and prepared for ceases to be an emergency."

## SOCIAL SERVICE ACTIVITIES

clothing and especially jewelry. Meantime some descriptions of women victims with special note of jewelry had been assembled with the hope that these descriptions might be checked with inquirers' descriptions and so avoid the painful necessity of having the relative view several victims in the process of identification. This proved helpful in a few cases. More accurate description of victims and better system might have made this more helpful. So far as possible visitors were accompanied by volunteers or Social Service on their visit to the improvised mortuary. Medical Social Service assisted at the mortuary where all inquirers were required to register. If identification was established the relative was accompanied to the Admitting Office. If not the next step was suggested. The address and telephone number of the Master File and other hospitals were given. By 5 p.m. Sunday identity of all but two men had been established.

At the request of the Admitting Officer one worker was assigned to telephone to some 60 patients who had appointments for admission during the ensuing week, explaining the necessity for postponement on account of the admission of disaster victims. Another worker was asked to call families of those dead whose homes were at a distance and explain the situation.

Our experience has convinced us that during a disaster no services can be considered mechanical. Giving information or compiling data and making records require a degree of judgment and skill not demanded under ordinary circumstances. Because of the extreme emotional tension under which people are suffering and the stunned sense of isolated bewilderment, instructions are often not readily comprehended. Customary routine such as asking for information needs to be individualized and far more skill in interviewing is required. Even the briefest contacts become charged with meaning and often the simplest services are valuable out of all proportion, such as dialing a telephone number for a confused relative.

On Sunday the morning following the disaster at the request of the Supervisor acting as head nurse in the special ward two medical social workers were assigned to the ward, one to attend the telephone line over which inquiries about patients were routed, the other to control admissions to the ward. This second assignment after 24 hours was carried by experienced volunteers from the War Service Committee. The assignment to the nurses' desk to answer telephone inquiries about patients rapidly developed into a fuller service. In cooperation with the head nurse an up-to-the-minute report on condition of the patients was maintained so that reports could be readily given to relatives. The patients at first too stunned to realize the situation soon began to ask questions. What had happened to wife or husband? Where were the others in the party? Had they escaped? Many patients had members of family and friends who were also victims. The living and dead members of groups were widely scattered. Messages of inquiry and reassurance passed rapidly to-and-fro. The information ac-

## IDA M CANNON

inquiries Being at the hospital entrance, it was here that a restless throng soon assembled Members of the press and police eager for lists of victims, representatives of army and navy seeking identification of their men, blood donors in great numbers responding to the urgent radio appeal One zealous young man brought in several groups of prospective donors he had enticed from passing street cars These people, at first directed to the Blood Bank Station, were later sifted by a responsible volunteer by questions of time of latest food and liquor intake

To get some idea of the pressure at the Information Desk, one must realize that while the doctors and nurses were absorbed in giving care to the living victims, and the Administration was wrestling with the problem of an improvised mortuary for the 75 dead, the community was being aroused to the magnitude of the tragedy by radio and swift traveling news Families and friends were setting out anxiously to locate some 650 missing persons

The listing and reporting of the 75 dead in the improvised mortuary were delayed partially awaiting authority from the Medical Examiner, and also because of the serious difficulty in identification The process of identification was shared by the police, Women's Defense Corps and various hospital personnel Most of the men had identification in their pockets These were listed and reported But the women, who were mostly in evening dress, were without handbags or coats Their clothing was often torn and burned Jewelry proved to be an important means of identification

In increasing numbers through the early morning and into Sunday afternoon the anxious relatives and friends came, some from considerable distance, singly, but more often in groups For some their visit here was the first effort to find the missing victim Some of them had already checked with the Master File and knew that there was no evidence that the one they sought was among the living Some had already made the rounds of the mortuaries The major task of the Social Service Department at the hospital entrance was meeting and interviewing these relatives and friends of those seeking victims Some 175 interviews were recorded In spite of extreme tension, shock and physical weariness these men and women acted with great dignity and restraint There was no hysteria In comment on the technic for handling the situation, we note that the inquirers awaiting interview were restless and needed to move about Many wanted to smoke and were not denied They were interviewed in turn by the Social Service Staff For the interview which served as some release of tension, they were seated and in a separate room, which gave some privacy The interview in introduction followed something of a pattern The name of the victim sought, relation to inquirer, a review of list of living Although in some instances this was a repetition of review at the Information Desk, another search for the familiar name was anxiously sought Hope lingered Then after explanation that there were unidentified dead we proceeded to get age, height, weight of victim, whether blonde or brunette, description of

## SOCIAL SERVICE ACTIVITIES

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cumulated was useful to the doctor in dealing with problems of when and how to inform the patient about the death or condition of other members of the family or party.

On direction of the Administrator on duty, incoming mail to patients was opened, read, and wherever there was question about reading certain messages to a patient, the approval of the doctor was secured. Many patients had bandages over their faces which prevented use of their eyes, and so it was important to read mail to them. Messages, letters and telegrams from patients were handled by Social Service for them.

Practical problems distressed patients and needed attention. Among these were witnessing a will, attending to insurance papers, inquiries about clothing and valuables, transfer of automobiles left near the scene of the disaster, job adjustments and communication with employers, letters to be written, household arrangements such as messages to maids at home, care of pets, liaison with army and navy authorities. Discrimination was necessary as to what could be handled by the social worker and what needed clinical or administrative sanction. There was need for some exercise of judgment and counseling also in relation to the patients' or families' requests which under the stress of the situation sometimes showed hasty ill-advised decisions.

On the third day, at the request of the staff doctors, the social worker on duty accompanied the doctor as he interviewed relatives who were still excluded from the ward. This interview served to establish in the visitors' minds the fact that the social worker was working with the doctor and that she could be used for interchange of messages and interpreting the patient's condition. A special attempt was made to relieve the emotional stress of the relatives by giving them an opportunity to talk. Some of them had special need for this. For instance, the father summoned from a distant city because of the imminent death of his daughter but arriving too late to clear up a misunderstanding between the daughter and her mother.

By previous agreement one of the Social Service Staff had been appointed liaison with the Red Cross Disaster Relief. Their resources had been promptly offered. Although most of our patients were in comfortable economic circumstances there were some serious and urgent needs for advice and guidance in meeting the necessary adjustments, especially for families at a distance. Daily intercommunication with the Red Cross was established early. Their generous outpouring of helpfulness material and friendly is another story.

Under the urgency of a disaster such as this the focus of clinical concern of the physician and nurse is sharpened, the area of attention markedly restricted. At the same time the personal and social aspects of the patients' problems are especially acute and distressing. For them the experience of sudden shift from well-being and gaiety to painful and serious injury, and for many the death of some loved ones, created deeply disturbing complica-

tions that needed special psychiatric attention. Deep grief experience came to many patients at a time when they were enduring physical suffering and immobilized and isolated they could not act for themselves. The necessary "no visitors" precaution made it more difficult to turn to their families for help.

Only by well integrated teamwork among all the professional personnel charged with the responsibility for service to patients could the total situation of each patient become comprehensible and be dealt with. This teamwork at the time of the disaster can be sustained and function only on a foundation of previous teamwork experience and mutual confidence. Thus Social Service was prepared to carry its own responsibilities and also some of the personal service to patients carried by nurses and doctors in the usual day's work. We are well aware that after all the various professional skills are expended in meeting the patients' acute needs there are left for many of them broken homes, responsibility for care of fatherless children and loneliness wounds which time and inner resources alone can heal.

## NEUROPSYCHIATRIC OBSERVATIONS

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THE STUDIES to be reported here do not deal with all the neuropsychiatric abnormalities which occurred on the Disaster Ward. Mild transient confusions and delirious conditions, fluctuations in consciousness, fleeting periods of restlessness were handled by surgeons and medical men without the need for a special psychiatric inquiry.

This report deals rather with the problems involved in the emotional adjustment of the patient to the disaster, with all its implications—disfigurement, lasting disability, loss of work, bereavement, and disturbed social situations. We wanted to learn how to recognize those patients who are liable to emotional disorders, to prevent such disorders if possible, and to help those who had become victims of untoward emotional reactions.

The first request for psychiatric help came through the social workers who were serving as liaison personnel with relatives and friends. They soon had become aware of the fact that the emotional upset which followed the discovery of a body had attained, in some of the relatives, the proportions of a major psychiatric condition and needed trained intervention, and it was at their insistence that we first witnessed the states of acute grief which will be discussed in detail later in this study.

From observing violent reactions in the relatives, we concluded that similar reactions might occur in the patients on the ward as soon as they were recovered enough to deal with the disruption of their social relationships.

The social workers continued to confer with the psychiatrist about the management of milder reactions and were extremely helpful in the arrangement of the subsequent systematic studies.

On the eighth day after the disaster, the psychiatrists were invited to review all patients still on the ward. The occasion was a dramatic psychotic episode in a woman who had not been confused and was not then showing signs of impairment of brain functions, but who had responded to the news of the death of her husband and son with a state of excitement and intense paranoid suspicions about the ward personnel. She also believed that nurses and doctors were considering her an immoral, sinful person and were plotting to detain her and to prepare for her punishment. She insisted upon leaving against the advice of the physicians and was able to persuade members of her family to demand her release. Psychiatric inquiry showed that this patient had had a former episode of mental abnormality with obsessive fears, depression, and mild agitation. Follow-up reports show that her subsequent adjustment has remained quite precarious with spurts of over-activity alternating with periods of apathy but that she has not developed any frank psychosis.

In the light of this incident it was decided to make a brief psychiatric study of all the patients left on the ward in order to be able to anticipate subsequent emotional disturbances. Seventeen patients have been so reviewed. Each received a neurologic and psychiatric examination. Abnormalities in mental status were recorded. A psychiatric history was obtained from the patient and with the help of social workers from the relatives. Plans then were made with surgeons, social workers and the occupational therapy workers for the best care of each patient in the light of our observations about his emotional reaction patterns and his former modes of adjustment.

The group as a whole was of fair intellectual level. Except in one case there was no aphasia or apraxia, and the disturbances of memory were limited to amnesic scotomata which was difficult to separate from the effects of impairment of consciousness at the time of the accident. The one case that showed neurologic symptoms with signs of cerebral lesions is described first.

One patient showed a clear-cut picture of cerebral lesion. This was a married woman, age 35, who was admitted in profound shock with stertorous breathing. She was quite red and carbon monoxide poisoning was diagnosed. At 11:30 P.M. oxygen was started and she had become active and noisy but was out of touch with her surroundings. Twelve hours later she was still restless and disoriented; she occasionally spoke short sentences that had little relation to the situation. By 7:00 P.M. she was extremely restless, thrashing about with her arms and legs and had to have restraint and sedation. After that she was quiet for 12 hours. During the next three days she alternated between periods of motor activity and periods of quiet due to sedatives. When aroused she thrashed about. On December 3 it was noted that the movements were definitely athetoid and were accompanied by facial grimaces. These movements reached an apex on the fifth to tenth days when she showed a full-blown picture of athetoid chorea, especially marked on the left side. Along with this there was jaundice, semicomatose and speechlessness.

The picture of athetoid chorea was gone by the fourteenth day. During the next two weeks the patient began speaking but showed marked aphasia which resolved into a dysarthria plus extensive memory defect (31st day). The memory improved over the next six weeks so that she could remember events for a number of hours and even up to three days. It is very much impaired even at the last examination (94th day); she remembers facts that she did certain things such as that she had lunch with a certain person; she seems to have lost all ability to revisualize to remember what the scenes were like and how people looked and even how objects looked. The dysarthria remains.

*Diagnosis*—There was evidently an extensive asphyxia of nerve cells of the brain. This appears to have been very widespread and to have affected both the basal ganglia and the cortex. In the cortex there is no special localization except that the lower end of the motor area in the left hemi-

puberty. These consist of trembling, heart palpitation, choking sensations, and the fear of impending disaster. She had not obtained any adequate help for her psychoneurotic symptoms and restricted her social activities and occupational plans because of them. She had come to consider her life a failure. The Coconut Grove incident had seemed to her the final fulfillment of all her fears. During her convalescent period in the hospital she was a very cooperative, if somewhat discouraged patient. Except for her timidity and anxiety, she had no emotional difficulties during her hospital convalescence, but after she left the hospital she was unable to make a satisfactory home adjustment. She feels too disfigured, believes she has no chance for further happiness, and is hopeless about the future. She is being continued in regular psychotherapeutic interviews.

The other patients did not show any significant history of previous psychiatric difficulties. None of them had positive symptoms of neurosis, psychosis, or personality defect.

#### REACTIONS TO BEREAVEMENT

Seven patients became problems of psychiatric study and management because their recovery was complicated by severe grief. This study provided an unusual opportunity to observe the mechanisms of grief by which the bereaved person reestablished his equilibrium after the loss of a beloved.

Within a few days after the incident, as soon as the patient recovered from the shock and clouded consciousness, the question arose of when to tell him about his loss. It was obvious that both the physical and mental condition must be such that the individual could tolerate the message.

Our observations concern seven bereaved patients. Three of them said that they had been told just at the right time. Three felt that they had been reasonably certain of the loss and the final confirmation appeared as a relief of uncertainty rather than as an additional shock. One patient suspended all inquiry about the details of her husband's fate for more than four weeks, deliberately occupying her thoughts with personal friends and pleasant fantasies and recollections. When, however, her relatives visited her, they became more and more uneasy because the range of topics discussed in conversation was necessarily small. Any reference to the lost person and any attempt at planning the future had to be avoided. It finally became the psychiatrist's task to confront the patient with the sad news. This was done in the slow process of gradually recalling to her the details of her family life, her relationships to her children and relatives, and making it inescapable for her to inquire positively about the fate of her husband. Her first reaction was to blame her relatives for withholding the news and in the subsequent interviews there was a marked hostility against the psychiatrist. After a grief period of less than a week she continued to make an unperturbed recovery so far as her physical condition was concerned. She has refused any further relationship with the psychiatrist but has apparently made a fairly good adjustment at home.

Frequent discussions between the surgeon and the psychiatrist were necessary to weigh somatic and psychologic factors bearing on the right moment of delivering the message of bereavement.

The second task of the psychiatrist was to assist the person with the adjustment to the loss and to steer him through the disturbing period of intense emotional upheaval which ensued during the subsequent weeks. It became apparent that the different patients showed considerable variation in their reactions. Common to all of them however was the following syndrome. Sensations of somatic distress occurring in waves lasting from 20 minutes to one hour: a feeling of tightness in the throat, choking with shortness of breath, need for sighing and an empty feeling in the abdomen, lack of power in the muscles and an intense subjective distress described as tension, lonesomeness or mental pain. The patient soon learned that these waves of discomfort could be precipitated by visits, by mentioning the deceased and by receiving sympathy. There was a tendency to avoid the syndrome at any cost, to refuse visits lest they would precipitate the reaction and to keep deliberately from one's thoughts all references to the deceased. Three men appeared in the psychiatric interviews to be in a state of tension with tightened facial musculature, unable to relax for fear they might 'break down'. It required considerable persuasion to yield to the grief process before they were willing to accept the discomfort of bereavement. One of the patients assumed a hostile attitude toward the psychiatrist, refusing to allow any references to the deceased and rather rudely asking him to leave. This attitude remained throughout his stay on the ward, and the prognosis for his condition is not good in the light of other observations. Hostility of this sort was encountered on only occasional visits with the other patients. They became willing to accept the grief process and to embark on a process of dealing in memory with the deceased person. As soon as this became possible there seemed to be a rapid relief of tension and the subsequent interviews were rather animated conversations in which the deceased was idealized and in which misgivings about the future adjustment were worked through.

It seems that the grieving person can delay his grieving period but not avoid it and that individuals who show no signs of grief during the period of convalescence from their somatic injuries are likely to have disabling disturbances at a later period. Prophylactic care is most important here. The patient must be allowed to carry through his grief reaction at the optimal time without undue delay; he must be assisted in his efforts to extricate himself from the bondage to the deceased, to be prepared to face the task of social readjustment when he leaves the hospital.

#### SPECIAL STUDIES

In addition to these problems of clinical management the solution of certain research problems has been brought nearer realization through the cooperation of a group of patients. For some time the Department of Psy-

## GRAPHIC REPRESENTATIONS OF ACTION-SILENCE

Figures 1 to 7 present interaction chronograms which show the striking differences in activity rates observed in patients with acute grief and in those suffering from other forms of morbid depression

Figures 2 and 3 represent mood disturbances which were seen in the Psychiatric Service. They showed depressive reactions with the usual slowness of response and underactivity. Figure 4 shows a patient who was found in a mild manic state of overactivity with euphoria and cheerful thought-content. Contrary to expectations, the interaction chronograms of bereaved patients as shown in Figures 5, 6 and 7 show overactivity and no retardation and slowness of response as seen in other depressed states

### SCHEMATIC REPRESENTATION OF TAPE, FIGS 1 TO 7 INCLUSIVE

To show actions, silences, double actions, and double silences (double actions and double silences recorded from Subject A)

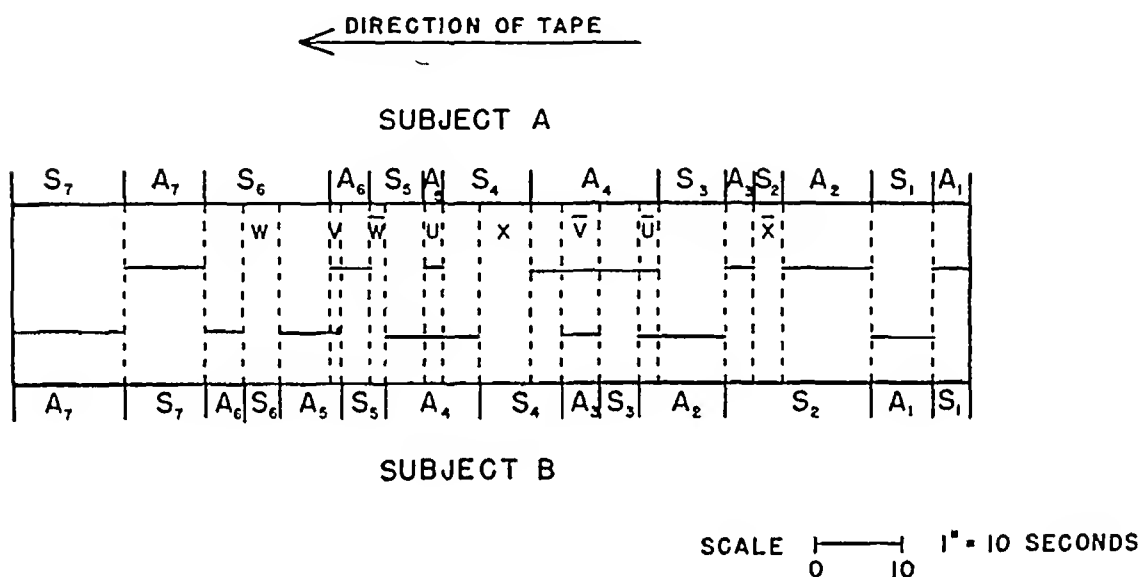


FIG 1 shows the various symbols assigned to different events in interaction (A)—activity (verbal or gestural), (S)—silence (inactivity) (u)—double action initiated by the patient (such as verbal interruption), (v)—double action initiated by the doctor, (w)—double silence due to failure to respond on part of the patient, (x)—double silence due to failure to respond on part of the doctor

A bar over the u, v, w, or x ( $\bar{u}$ ) indicates double actions or double silences after which the patient continues

The tape moves at a speed of five inches per minute. An observer sits behind the screen and observes through a one way window. She records on the moving tape by means of keys the activities of the two participants in the interview under the headings mentioned above. This tape is then mathematically analyzed to give the curves shown in Figures 2 to 7

Figures 2, 3, 4, 5, 6 and 7 are graphic presentations in "cumulative series" of four types of relationships

(1) A-S represents the relationship of the patient's periods of activity to his periods of silence. The more overactive he is the more positive is the slope of the A-S curve, the more underactive he is the more negative is the slope of this curve

(2) u-w represents the relationship of the patient's double actions (such as his interruptions of the doctor) to his failure to respond

(3) v-x represents the relationship of the doctor's double actions (such as his interruptions of the patient) to his failure to respond

(4)  $\theta$ -o represents the relationship of the patient's initiations of actions to those of the doctor

# CURVES IN ACUTE GRIEF AND ALLIED CONDITIONS

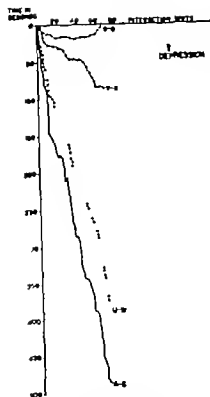


FIG. 2—A morbid depression of mood not complicated by grief. Note the steep negative slope of the A-S curve and the U-W curve. The deficit of action in relationship to silence on the part of the patient is 450 seconds in a 40-minute interview.



FIG. 3—A milder depression. The steep positive slope of the A-S curve is interrupted by occasional periods of increased activity but there is still a marked deficit in action.

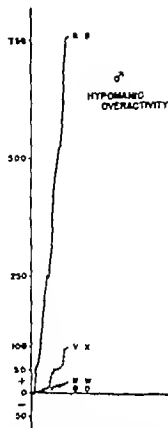


FIG. 4—A patient in mild manic episode with marked overactivity. The surplus of activity over silence in a 40-minute interview is 750 seconds.



FIG. 5—Acute grief reaction. The patient appears depressed during the interview but shows considerable overactivity. The A-S curve has a steep positive slope. The surplus of action over silence in a 40-minute interview is 610 seconds.



FIG. 6—Acute grief reaction with strongly depressive thought content. Note the markedly positive slope of the A-S curve. The curve resembles that of Figure 4. There is a marked surplus of activity over silence.

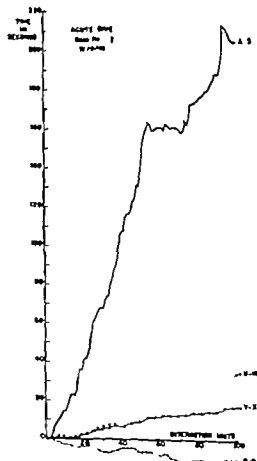


FIG. 7—Acute grief reaction with less pronounced overactivity.



chiatry has been interested in the physiologic and psychologic aspects of acute grief. Since acute grief is one of the most frequent psychogenic factors found in patients with psychosomatic disorders, such as asthma, colitis, and rheumatoid arthritis, we have been anxious to discover what physiologic features of grief might play a rôle in contributing to the etiology of these disorders.

In the fire victims there was evidence of disturbances in autonomic functions. The pupils were generally large. During the surges of acute grief described above, there was usually sighing respiration, "hot waves" to the head, flushed face and perspiration. Systematic spiograms were not satisfactory because of the chest involvement. We have, however, been able to carry on observations in bereaved relatives who showed the same sighing respiration. The result of these studies will be reported elsewhere. There is indication that the altered respiratory activity, combined with the disturbance of sleep and appetite, may form the nucleus of a physiologic disturbance which forms the background for the "emotional distress" described by the patients.

Our data are somewhat more complete for the study of the amount of activity presented by the fire victims and bereaved patients. It is known that in states of morbid depression a patient is likely to be "retarded" in speech and action. Contrary to expectation, in the state of depression and unhappiness following such a disaster experience, there is not a reduction in activity as is seen in cases of psychotic depression, instead there is an increase in need for activity. This can be strikingly demonstrated by a new measuring device, the "interaction chronogram." The patients were examined during a psychiatric interview for the timing of their verbal and gestural activity. A record was made in this way of their interaction with the psychiatrist and a graphic presentation was furnished, showing the balance of activity and inactivity at any given time. These graphs furnish an objective record of the patient's capacity to be active, of his hesitations after questions, and of his tendency to "out-talk" the examiner in conversation. We found that all bereaved persons examined, showed a positive slope of the action-silence curve, indicating a surplus of activity over inactivity (Figs 1-7) (For a brief description of procedure see Figure 1, for a complete description ref. Chapple and Lindemann<sup>1</sup>).

This finding is of special significance because it indicates a drive for activity in individuals who at the same time complain about apathy, inability to initiate any action, and lack of interest in their ordinary pursuits. Our observations seem to indicate that there is a good deal of drive for activity and the lack of "conduct patterns" by which to express their drive. A good many daily activities were conditioned to the presence of the deceased and could no longer operate. But more than that, other activities not obviously connected with the presence of the deceased have lost their meaning and are carried out only with difficulty. It is therefore, not surprising that only two of our series of mourning individuals were able to resume their ordinary

## NEUROPSYCHIATRIC OBSERVATIONS

activities after leaving the hospital. The others still find themselves 'unlucky' in initiative and looking to others for suggestions to follow.

These observations are still going on but already indicate the necessity of a careful follow up study of both patients and relatives. We have made an effort to reach as many individuals involved in the tragedy as possible but several months must elapse before any final conclusions can be drawn.

**Discussion.**—Of 39 patients admitted to the hospital seven died within 62 hours. Of the survivors at least 14 presented neuropsychiatric problems. This high incidence may seem surprising but it fits well with the experiences of psychiatrists working in general hospitals. Forty five to 55 per cent of the patient population are likely to present psychologic factors in their problems.

Unless the psychiatrist has an opportunity to see all the victims of a disaster danger signals and opportunities for help along psychologic lines may be overlooked since they are by no means obvious to the untrained worker.

Conditions predominantly due to cerebral damage were rare, probably because they usually lead to death. Conditions predominantly due to psychogenic factors showed a high incidence. In all patients with clear-cut neuroses and psychoses the psychiatric history offered clues as to the likelihood of such development under stress. This observation fits well with recent studies concerning traumatic neuroses in the armed forces.<sup>2, 3</sup> It seems well founded that induction boards refuse admission to the armed forces to candidates who show a former history of psychosis or psychoneurosis.

The more severe emotional disturbances encountered in formerly well adjusted patients seemed to be due not so much to the impersonal effects of the disaster (fright and horror) as to the problems in personal and social relationships involving conflict and guilt. Similar observations are reported by Sargant,<sup>4</sup> in 1940 and Wilson<sup>5</sup> in 1941 after the disaster experiences of members of the British armed forces and civilian population.

Psychiatric assistance in the solution of these personal problems and in readjustment after a social crisis forms an important part of the care of disaster victims.

### SUGGESTIONS FOR FUTURE EMERGENCIES

Our observations seem to indicate that the psychiatrist can operate as a useful member of a disaster unit. His work may be divided into three phases.

In the first few days severe shock and life saving procedures occupy the field. Apathy and excitement confusion and delirious states have to be handled by proper sedation and proper surroundings. In our present observations we have only indirect evidence of the victims' emotional states at that time. Two patients complained of the lack of a chance for enduring contact with one person—doctor attendant or nurse everything seemed to change every person who arrived seemed to be new no information or outside news

was available, the days were ones of utter bewilderment, offering no frame of reference. It might be advisable to have the ward personnel as small as circumstances permit or so divided that patients have a chance to deal with the same person repeatedly.

The second phase deals with the psychiatric care of the convalescent patient, advising him in his transitory problems, determining when messages should be delivered or revelations made, and managing with the patient his efforts to readjust.

The third phase deals with the psychiatric care of the convalescent patient after he leaves the hospital and his proper readjustment in the community. We can, in this manner, have reasonable hope of preventing the occurrence of prolonged maladjustment or traumatic neurosis.

During the first phase, the psychiatrist's chief contribution is his aid to the relatives and his counsel to the medical social worker who is dealing with the numerous problems of family and work relationships. During the second, he is intimately involved with the internist and surgeon and must continue his contact with the social worker, which becomes even more important during the third, when social readjustment forms the center of interest. Throughout the whole of the three periods, not the least of the psychiatrist's responsibilities is determining what can safely be delegated to the medical social worker and guiding her in her efforts.

It seems fair to conclude that it is desirable to have psychiatric evaluation of patients early in the course of their hospital care, continued psychiatric attention to those patients who are in a precarious emotional state, and, lastly, aid in making readjustment, especially to bereavement, after leaving the hospital.

#### REFERENCES

- <sup>1</sup> Chapple, E. D., and Lindemann, E. Clinical Implications of Measurements of Interaction Rates in Psychiatric Interviews. *Applied Anthropology*, 1, 1-11, 1942.
- <sup>2</sup> Ross, T. A. *Lectures in War Neuroses*. Williams and Wilkins, Baltimore, 1941.
- <sup>3</sup> Gillespie, R. D. *Psychological Effects of War on Citizen and Soldier*. Norton, New York, 1942.
- <sup>4</sup> Sargant, W., Slater, E. Acute War Neuroses, *Lancet*, 2, 1, 1940.
- <sup>5</sup> Wilson, A. T. M. Reactive Emotional Disorders. *Practitioner*, 146, 254-258, 1941.

# RESUSCITATION AND SEDATION OF PATIENTS WITH BURNS WHICH INCLUDE THE AIRWAY

## SOME PROBLEMS OF IMMEDIATE THERAPY

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CERTAIN ASPECTS of the Coconut Grove disaster are characteristic of conditions encountered in most conflagrations of the flash burn type. In this report attention will be given primarily to the factors of general interest. These matters are particularly appropriate for consideration at this time for in the present widespread use of mechanized warfare flash burns are exceedingly common.

As the patients from the scene of the disaster were crowded into the hospital it became apparent early that they were divided sharply into two groups. The living and the dead or near dead. None in the former group died in the first 12 hours; none in the latter group lived more than a few minutes after arrival.

The patients who lay quietly at rest on arrival were in the minority. As soon as it could be established tentatively that these few individuals had not suffered central nervous system injuries or were not stupefied by smoke inhalation and showed no signs of approaching shock, it was clear that the larger hyperactive group needed the most attention although at the same time the importance of watching these quiet ones was not minimized.

An outstanding characteristic of the living group was hyperactivity, even to the extent of mania in some cases. One's first impulse is to assume that this hyperactivity is due to pain. From the history of other similar tragedies this appears to have been the usual assumption and the patients treated accordingly. A careful appraisal of the causes of this hyperactivity is of real assistance in planning individual therapy. In the Coconut Grove disaster we had a unique opportunity for simultaneous observation of a considerable number (39) of victims of the same accident. It was quite apparent that pain was an improbable cause of the observed hyperactivity in many cases. *As time progressed the importance of correctly diagnosing the cause of this hyperactivity in a given case became increasingly clear for proper therapy depended upon differentiating between three possible major causes.*

### COMMON CAUSES OF HYPERACTIVITY

(1) *Pain*.—Unquestionably pain was present in many patients. This was due to burns, to irritation of mucous membranes chiefly the eyes and the airway by irritant gases and to physical violence as a result of the panic that had occurred.

(2) *Fear and Hysteria*.—as a result of the individual's experiences appeared

to influence the behavior observed of those who had no injuries and doubtless were also a factor in the behavior of many of those with physical injuries as well. It will be recalled that this disaster occurred in a night club at about 10 15 P M. Alcohol in some cases doubtless contributed to the excitement and the lack of self-control. The panic of the crowd and the physical pain and discomfort shared in producing the hysteria observed.

(3) *Anoxia*—Cerebral anoxia is well known to give rise to excitement, occasionally to loss of self-control, and at times to manic behavior. This sequence is not infrequently encountered in chronic heart failure. Anoxia was probably a factor in our patients due to two main causes. (1) Interference with oxygen intake from obstruction of the airway was caused by a number of factors, for example, foreign bodies, chiefly vomitus. (This was a common hazard in the comatose but not in the hyperactive group.) Severe bronchial spasm occurred from pulmonary irritation caused by inspiration of the hot and noxious gases and probably interfered with the intake of oxygen. Edema of the airway developed into a problem beginning chiefly about four hours following the burns. How much of a lethal factor edema of the airway was in those who lived only a few minutes, is uncertain. (2) Impairment of oxygen transport by the blood was a factor in the development of anoxia largely as the result of the formation of carbon monoxide hemoglobin. Neither methemoglobin formation nor hemolysis as the result of encounter with noxious gases was a factor in our cases. While acute anemia must always be considered as a cause of cerebral anoxia, neither hemorrhage from associated wounds nor low blood pressure from shock was important here. In about one-third of the patients measurement of the blood pressure was not carried out because of interfering burned areas. Judging from other clinical signs these patients were not in shock. Where it was possible to measure blood pressure two patients were found to have brief periods of hypotension, but frank surgical shock did not develop in any of the 39 patients.

#### THERAPY OF THE CAUSES OF HYPERACTIVITY

The importance of properly diagnosing the cause of the hyperactivity emerges from considerations of therapy. Moreover a clue arises here as to the reasons for the often made recommendation of the use of enormous doses of morphine in burned patients. Occasionally large doses may be necessary, it appears probable in many cases that they are not only unnecessary but are in fact contraindicated. When the hyperactivity of the patients is caused by fear or hysteria or by cerebral anoxia the use of large doses of morphine is obviously unwise. *It seems probable that in burned patients morphine may often have been used in an attempt to treat conditions which will not respond favorably to morphine however large the dose.* An examination of the probable reasons for the use of large doses of morphine may throw some light on what is rational sedation for this group of patients.

## IMMEDIATE THERAPY AFTER BURNS

### PAIN

It has come to be accepted as a fact in medical practice that enormous doses of morphine must be used in the treatment of burned patients doses that under normal circumstances might of themselves be fatal. Against this is the observation (Hardy, Wolff and Goodell<sup>1</sup>) that the analgetic action of morphine increases rapidly up to 10 mg ( $\frac{1}{8}$  grain) intramuscularly but is increased little by doubling or tripling the dose. While little of added benefit is obtained by thus doubling or tripling great increase in the toxic effects particularly respiratory depression results therefrom. It seems reasonable to question the advisability of the use of the customary large doses of morphine. Support for this view also emerges as will be described below, from the realization that other factors besides pain may help to account for the hyperactive, even manic behavior of individuals who have been subjected to a conflagration.

For pain morphine administration is the treatment of choice. It must constantly be borne in mind that the common tendency in a disaster of this kind is to overmedicate. Safety depends upon the use of rather small divided doses repeated as necessary. Emphasis on these elementary matters may seem needless but our experience was otherwise. For intravenous administration somewhat smaller doses are chosen than for subcutaneous or intramuscular use. Intravenous injection of the morphine is best—8 to 10 mg ( $\frac{1}{8}$  to  $\frac{1}{6}$  gr.) doses are used. Such doses as these should be injected over 15 to 30 seconds and may be repeated intravenously in about 15 minutes until the desired effect is obtained. When many patients need treatment at once there will often not be time to administer the agent intravenously.

Whenever the subcutaneous or intramuscular routes of administration of morphine (or other agents) are considered it must be borne in mind that under circumstances where the peripheral circulation is slow or inactive the injected agent may not be absorbed. In patients coming from a fire several conditions tend to reduce the peripheral circulation and consequently the rate of absorption of agents injected into the subcutaneous or intramuscular regions. Chilling from cold water spray and water soaked clothing (our patients had in some cases rectal temperatures as low as 94° F. pain and fear and low blood pressure from various causes. Under circumstances such as these agents injected into the subcutaneous or intramuscular regions will be absorbed very slowly if at all. Lack of attention to this possibility may result in repeated injection of the agent into these "refractory" patients. Later when the peripheral circulation has been reestablished by shock therapy or warmth the total injected dose may be absorbed at once with disastrous results.

When large numbers are to be cared for as quickly as possible when the peripheral veins are collapsed when slow absorption of the agent is desired and for various other reasons subcutaneous or intramuscular use may be employed. Here 15 mg ( $\frac{3}{8}$  gr.) doses may be administered. In

such a case it is advisable to make up the solution (when a considerable number of patients must be treated rapidly) in a 20 cc syringe with 15 mg ( $\frac{1}{4}$  gr) per cc concentration. A second such dose can be repeated in 20 minutes for a robust subject with severe burns. Increase in morphine medication beyond 30 mg ( $\frac{1}{2}$  gr) is made only after one has assured himself that the need is for treatment of pain rather than fear or hysteria or anoxia. Even then, the justification in most cases for such large doses is questionable, as mentioned above.

If there is any possibility that large doses of morphine will be required it is advisable to use an extremity for their injection, and the site should be marked with a dye so that if signs of overmorphinization appear, absorption can be delayed by the use of a tourniquet above the site of injection. An unburned extremity should of course be chosen, for a tourniquet placed about one with peripheral burns would increase the edema formation in the injured area.

#### FEAR AND HYSTERIA

Fear and hysteria are best treated by repeated intravenous administration of a barbiturate, for example, sodium pentobarbital (nembutal) in 90 mg (gr 15) doses. In patients with pulmonary damage it is doubtful if more than two such doses should be given initially. While opinions are divided as to the wisdom of using paraldehyde in patients with injury to the lungs, there appears to be no serious objection to the use of small doses intravenously, as follows. Two or three cubic centimeters of paraldehyde may be injected over a half minute. One patient (Case 7) received paraldehyde, 4 cc intravenously.

#### ANOXIA FROM AN INADEQUATE AIRWAY

Treatment of an inadequate airway takes precedence over all other forms of therapy. The following factors require consideration in this therapy.

*The Removal of Foreign Material*—The Coconut Grove victims were either dining or had only recently finished dinner. Probably vomiting was more frequently encountered in this group than is usually the case in burned patients. Vomitus in the airway of the patients who arrived at the hospital either dead or in a moribund condition may have hastened some deaths. It is unlikely that such obstruction was present in the other, the hyperactive, group of patients. Occasionally, aspiration of the mouth and throat of these patients was carried out as a preventive measure.

*Intratracheal intubation* was carried out in three patients. In one of these cases it was necessary about two hours after admission.

It was considered to be life-saving in this case. Gross overdosage with morphine was present, and the intratracheal tube facilitated artificial respiration which was necessary intermittently over a five-hour period. In another case intratracheal intubation was used to facilitate bronchial aspiration several days after the accident. In the third case the procedure was used terminally to facilitate respiration preceding death. One or two other patients would have

## IMMEDIATE THERAPY AFTER BURNS

received intratracheal intubation had they not been vomiting. It is usually unwise to introduce an intratracheal tube into a vomiting patient. For the necessary local anesthesia may permit the aspiration of vomitus. It is also unwise to insert an intratracheal tube surrounded by an inflatable cuff to prevent aspiration, for such inflatable cuffs have produced damage to normal mucosa of such degree that subsequent to their removal fatal local edema has occurred; therefore we did not choose to use them in these subjects with already inflamed tissues. The best solution of this problem seems to be careful watching of all patients with immediate tracheotomy in those that are vomiting when the airway shows signs of inadequacy.

*Tracheotomy* was required for the first time six and one half hours following the fire. In all five tracheotomies were carried out in the 39 patients during the recovery period. Three of the five patients died. The onset of serious edema of the airway in our cases several hours after the burns is in agreement with the history of other similar disasters notably that of the Crile Clinic.

*Treatment of Bronchospasm*—How great a role bronchospasm plays in the inadequate ventilation of the lungs in such patients is difficult to estimate. The bronchospasm was initiated presumably by heat or by the irritant gases breathed. It may have been a factor in precipitating or aggravating pulmonary edema formation. It was the consensus of opinion of those who examined the chests and roentgenograms of the patients that bronchospasm was a factor in producing the well-demonstrated peripheral trapping of air. Local edema as well as foreign bodies arising from sloughs in the bronchiolar walls were also doubtless involved in this. Attempts to treat this bronchospasm with epinephrine or ephedrine appeared to be quite unsuccessful. In a few cases the intravenous administration of 0.5 Gm (7.5 grs.) of aminophylline appeared to be followed immediately by better ventilation and in some cases by cough with the raising of sputum. This benefit may have lasted for only ten or fifteen minutes; estimation of this was difficult. Whenever injury of the airway has occurred as in these patients it is important to humidify the air breathed. All gases administered should be saturated with water vapor.

*Oxygen Inspired*—Patients showing any signs of anoxia were immediately given by mask 100 per cent oxygen to breathe. In the first six hours seven of 39 patients required high oxygen concentrations. These were administered in order to get not only as full saturation of hemoglobin as possible through the damaged respiratory epithelium but also to get the advantage of oxygen dissolved in the blood plasma. Subsequently a total of 13 patients required oxygen therapy chiefly by tent.

*Increased Pressure in the Airway*—When oxygen is administered in a closed system under positive pressure a greater diffusing surface is afforded the alveolar gases and the blood and possibly the smaller airways are increased in diameter by the pressure, with the result that obstructing secretions are less effective in blocking the passages than they were. If this is the



situation, drainage might also be promoted by positive pressure. It is said that the use of positive pressure will prevent or curtail the formation of pulmonary edema. This seems to be open to question. Some believe, although incorrectly, that the partial pressure of the alveolar oxygen can be significantly increased by safe positive pressures. While this might be true at very high altitudes it is not true at ordinary atmospheric pressures. Notwithstanding the possible advantages to be gained from positive pressure we decided, rightly or wrongly, not to use it in these cases. In the first place, in a fairly wide experience with positive pressure in patients undergoing thoracic surgery it has been our observation that this procedure often lowers the systemic arterial pressure, probably by interfering with the passage of the blood through stretched out and narrowed alveolar vessels with the result that filling of the left heart is impeded. Positive pressure appears to interfere with carbon dioxide elimination. Finally, several patients exhibited a paradoxical pulse. We construed this to be a further argument against the use of positive pressure.

*Helium*—When the tidal volume of air is normal or near normal it is unlikely that helium will be of value as a vehicle for oxygen, although it might be argued that if some bronchospasm is present the use of helium might be desirable. Our experience with helium in these cases was limited to a few trials of an experimental nature in which 75 per cent helium with 25 per cent oxygen was compared with 100 per cent oxygen. It was not possible at this time to make careful blood gas studies. We were not able to detect any improvement in the skin blood color with the helium and oxygen mixture as opposed to the high oxygen atmosphere. On the other hand, the pulse rates under the latter atmosphere were about 20 beats slower than when the helium and oxygen mixture was used (about 140 against 160). The difference in pulse rates suggests that oxygenation was better when 100 per cent oxygen was used than when the helium was employed.

#### ANOXIA FROM INADEQUATE TRANSPORT OF OXYGEN BY THE BLOOD

*Carbon Monoxide Poisoning*—While many of the dead patients showed signs of carbon monoxide poisoning only two, questionably three, of our 39 living patients showed fairly definite signs of it. In these, attempts were made to eliminate the carbon monoxide by the administration of a continuous stream of oxygen containing five to seven per cent carbon dioxide. No rebreathing was permitted here. It is as desirable to give whole blood as soon as possible to these patients as it is to patients who may be anemic following hemorrhage or anemic from encounters with hemolytic gases in the smoke breathed.<sup>2</sup>

*Shock*—When the patients arrived we supposed, incorrectly, that many cases of shock would develop. To combat shock, the intravenous injection of fluid was started on each patient within 15 minutes of the time of his arrival, in order to expedite the use of plasma as soon as it could be made ready. Both physiologic saline and five per cent glucose solutions were

## IMMEDIATE THERAPY AFTER BURNS

used. The volume of these fluids administered was sharply restricted. From 200 to 500 cc. were administered before plasma was started or until the decision was made that intravenous fluids were not necessary. Twenty nine patients received an average of 4.2 units (250 cc. unit) of plasma apiece in the first 24 hours. The variation was from one to nine units per individual in the first 24 hours. In the first seven days 147 units of plasma were administered. Also in the first seven days 16 whole blood transfusions were administered for patients with reduced oxygen capacity of their blood.

As already pointed out none of our patients developed frank shock. In the two instances in our cases where the blood pressure was low even for a brief period the head-down position was used but as soon as the systolic arterial pressure had risen to 80 mm. Hg we began gradually to reverse the position from head-down to head up. Damage to the lungs must be assumed in patients such as these from the Coconut Grove even though it is not apparent. The head up position reduces the pulmonary venous pressure and minimizes the tendency to edema formation in the lungs.

### OBSERVATION OF PATIENTS FOLLOWING IMMEDIATE THERAPY

Factors of importance in the medical administration and organization of the treatment of large numbers of wounded individuals have been dealt with by Dr. Faxon in his accompanying article, and elsewhere by Faxon and Churchill.<sup>2</sup> An indispensable part of the therapy of patients with burns of the airway is continuous and prolonged watchfulness of the respiratory and circulatory systems as well as of the patient's comfort. In the Coconut Grove disaster this was handled by the following personnel for dealing with our 39 patients:

Two physicians made rapid and continuous chest rounds on all patients following the initial treatment. It was their responsibility to watch the pulmonary ventilation with particular attention to the development of pulmonary edema and to inadequate oxygenation of the blood from any cause. They called attention to deteriorating cases. They requested any new therapy needed as intubation, oxygen therapy, etc.

Two men (medical students) made continuous rounds, determined blood pressures, pulse rates and recorded these data.

One physician made "medication rounds" constantly looking for patients who needed further drug therapy, diagnosed the need and administered the appropriate agent. Constant watchfulness for overmedication is essential.

### DELAYED REACTIONS

Delayed reactions are to be anticipated. Constant alertness must be maintained for the signs of (a) overmedication particularly in cases where morphine may have been administered subcutaneously with delayed absorption as in patients with poor peripheral circulation as a result of chilling or shock; (b) shock, as a result of plasma loss from burned surfaces or other cause; (c) oropharyngeal, tracheal or pulmonary edema; and (d) central nervous

system damage with delayed onset of cerebral edema with increased intracranial pressure

Observations of the condition of the blood is of great help in guiding the care of these patients and in providing accurate quantitative data concerning the trend of delayed reactions. For example, the increased time required for a blanched area (made by the light pressure of a finger on the skin of the forehead) to fill in, often provides, in demonstrating the slowing of the peripheral circulation, a sharp warning of decline in the patient's circulatory condition, and of the possible approach of shock. The color of the blood must be maintained as near normal as possible. If assistants and equipment are available, hematocrit readings are obtained, for they are of great value as a guide to the need for whole blood or plasma. These were available by four and one-half hours (3 A M) following the accident. The determination of the plasma protein level refractometrically is a brief, simple procedure of value. In the days following the disaster more elaborate studies were possible. Helpful here were determinations (in arterial blood) of oxygen content and capacity. In one or two cases low oxygen values showed an urgent need for red cells. This might have been surmised some hours earlier had adequate attention been given to the low hematocrit values. Carbon dioxide content, plasma  $p_H$ , and plasma electrolyte values were helpful although not as important as the previously mentioned determinations.

#### NEED FOR FUTURE WORK

The Cocoanut Grove disaster called attention to the lack of information concerning the pulmonary lesions produced by fires and at the same time emphasized the need for study of this problem. For many years it has been known that pulmonary burns produced delayed effects in that, initially, victims appear to be in good condition and then rather suddenly develop respiratory impairment, obstruction, possibly bronchospasm and edema, and die. This was strikingly illustrated in the Crile Clinic disaster at Cleveland several years ago, and again recently here. Important gaps in therapeutic knowledge are concerned with (a) how best to overcome deficient gaseous exchange in the lungs arising from bronchospasm or caused by edema and by tissue sloughs, and (b) how to prevent these conditions. With the increase in flash-burns as a result of mechanized warfare, or for that matter mechanized civilization, therapy of the pulmonary lesions involved urgently needs study.

#### SUMMARY AND CONCLUSIONS

The patients who survived the Cocoanut Grove disaster long enough to receive therapy were in many cases hyperactive, even manic. Proper therapy depended upon correctly diagnosing the cause of this hyperactivity in a given case. Three major causes were Pain, fear and hysteria, and cerebral anoxia.

Morphine is a useful therapeutic agent only for those in the first of these three groups. In the other two groups it is not only ineffective but is contraindicated in large doses. Although large doses of morphine are often

## IMMEDIATE THERAPY AFTER BURNS

employed in treating patients from a conflagration it seems probable that morphine may often have been used in an attempt to treat conditions which will not respond favorably to morphine irrespective of how large the dose.

In patients who have been water soaked and chilled who are frightened or who are approaching shock or whose peripheral circulation is otherwise greatly reduced it is unwise to administer morphine (or other agents) subcutaneously or intramuscularly for absorption will be either absent or greatly retarded. Lack of effect may lead to repeated administration of the agent in an effort to obtain an effect. Later when the circulation has improved the total of the subcutaneous injection may enter the circulation at one time with serious even fatal consequences. Morphine should be administered intravenously to such patients. If because of the great number of patients to be cared for one cannot take time for intravenous administration of morphine, the agent should be injected into an unburned extremity and the injection site marked with ink or a dye so that if too great absorption of the agent is apparent later on the inflow can be checked by means of a tourniquet.

For fear and hysteria, intravenously administered barbiturates are useful. For anoxia, arising chiefly from carbon monoxide poisoning the treatment is seven per cent carbon dioxide in 93 per cent oxygen in continuous stream (without rebreathing) and with the administration of whole blood.

Various oxygen therapy techniques (intratracheal intubation tracheotomy, helium positive pressure) are considered and reasons offered for discarding or employing them in treating the anoxia. The oxygenation problem is greatly complicated by severe bronchospasm and pulmonary edema. Consideration of these factors leads to a discussion of needs for future work.

## REFERENCES

- <sup>1</sup> Hardy J. D., Wolff H. G., and Goodell H. Studies on Pain. The Analgesic Action of Morphine and Codeine in Man. *Am. J. Physiol.*, 129, 375, 1940.
- <sup>2</sup> Drinker C. K. Carbon Monoxide Asphyxia. Oxford University Press, New York, 1938.
- <sup>3</sup> Faxon N. W., and Churchill E. D. The Coconut Grove Disaster in Boston. *J. A. M. A.* 120 1385-1388, 1942.

# THE PULMONARY COMPLICATIONS A CLINICAL DESCRIPTION

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MANY BURNED patients have pulmonary lesions due to thermal or chemical burns of the lung, and an important part of burn management consists in the recognition and proper treatment of these pulmonary burns. This was particularly well emphasized in the Cocoanut Grove cases treated at this hospital, since all seven of the deaths were due to pulmonary complications. In fact, only three of the 39 patients were wholly free from respiratory symptoms, and they had covered their mouths with wet cloths or some article of clothing. A wet handkerchief appeared to have afforded adequate protection in one individual. There was little correlation between the severity of surface burns and the extent of pulmonary damage, and it was, therefore, necessary to watch for pulmonary signs even in those who were only slightly burned.

The first clue to the high incidence of pulmonary burns was afforded by the number who died within the first few minutes after reaching the hospital. They were very cyanotic, comatose or restless, and had severe upper respiratory damage. The surviving patients on arrival showed varying degrees of restlessness or excitement but soon became quiet following medication and removal to the ward. None was very cyanotic at this time, some were cherry-red in color, suggesting carbon monoxide inhalation. Most of them were burned about the mouth and nose, with singed nasal hair and reddening of the nasal mucous membranes. In general, the patients during the first three hours were breathing quietly and superficially, and coughing weakly. Chest examination at first showed distant breath sounds and this was associated with scattered basal râles in many cases. Several of the more severely burned victims were delirious, and two of these were quieted promptly by oxygen inhalation, suggesting that the delirium was due to anoxia aggravated by carbon monoxide poisoning and slowing of the respirations from morphine. Several patients, notably Case 25, soon became dyspneic, and progressed to a critical condition within a few hours.

About three hours after the fire, dyspnea suddenly appeared in others associated with cyanosis, restlessness, and increased râles. Since there was a rapid accumulation of edema in the external burns at this time, it is probable that burned pulmonary areas were undergoing similar changes. Thus, Case 25, who had shown early dyspnea but had remained quiet during the first three hours, became so short of breath that he insisted on getting on his hands and knees to facilitate breathing. An oxygen tent promptly

relieved cyanosis but had no effect on the dyspnea. Complications in this stage were (1) acute dilatation of the stomach in two of the most severely dyspneic patients (2) a wildly delirious state, apparently due to anoxia (3) auricular fibrillation in Case 2 eventually relieved by oxygen administration. This period of dyspnea subsided within a few hours.

A more critical period occurred about 24 hours after the fire and continued for the next 36. Dyspnea and cyanosis became much aggravated in certain patients and rales again spread. This fulminating state was obviously due to edema from burns of the upper air passages, trachea and bronchial tree. Laryngeal examination had demonstrated reddening, edema, and burned areas extending beyond the vocal cords. Because of the critical nature of symptoms at this time radical therapy appeared indicated and intubations and tracheotomies were done in several instances. Only two of the five treated by tracheotomy survived. In all, seven patients died. Necropsies on three indicated that the lesions had been too widespread to be relieved by these procedures even though additional oxygen was fed by catheter through the tracheal cannula. It is clear that an accurate estimate of the extent of a pulmonary burn cannot be made soon after occurrence but it would appear that tracheotomy or tracheal intubation is indicated since it affords a chance of reducing the labor of respiration in a weakened patient by facilitating the passage of oxygen to the alveoli.

Whether phosgene or nitrous fumes were present in the smoke as might be suspected from the delayed edema apparently must remain a matter for speculation. Several of the patients exhibiting symptoms (Cases 5, 6 and 19) were exposed only to fumes and heat, and those for but a short period and did not come in contact with flame at all. Case 13, on the other hand, had severe face and nasal burns with denudation of her lower turbinate bones but developed only slight lung complications. Professor Alan Moritz found carbon monoxide in a high proportion of those dead on arrival at the hospital, but no methemoglobin or porphyrin in the blood of the one patient tested. He is of the opinion that oxides of nitrogen were in the smoke, and it is noteworthy that the pattern of pulmonary reaction in the Coconut Grove survivors was not unlike that of the Cleveland Clinic fire.

Following subsidence of the epidemic like attack of pulmonary edema, the final subacute stage of pulmonary manifestations set in of which the pathologic basis was diffuse bronchiolitis. This resulted in (1) obstruction of the air passages particularly in the bases sufficient to produce localized lobular collapse and (2) trapping of air at the apices with acute emphysema. Both gave characteristic physical signs: the percussion note was highly resonant over emphysematous areas and normal or slightly dull over the small areas of collapse while the most striking finding was the surprisingly diminished breath sounds over the entire chest, most evident in the lower portions. Bronchial breathing was not heard except in Case 6 and at one time in Case 27. Rales remarkably few in number were fine and crackling in the first few days and later coarse in character.

The absence of bronchial breathing suggested that the bronchi were plugged with secretion which prevented bronchial breath sounds from being transmitted to the periphery. This interpretation was borne out in many of the patients when the usual physical signs of pulmonary collapse disappeared after the coughing up of mucus and when similar signs appeared elsewhere. Thus, Case 20 had typical signs of collapse of the left lower lobe on the second day which disappeared on the next day after she coughed up a plug of pinkish-black material the shape of a medium-sized bronchus. On the fourth day, massive collapse of the other side was found to be present. The next day both lungs had cleared considerably, followed by appearance in the sputum of numerous brown plugs of mucus. A similar sequence occurred in Case 27 (who died on the third day). Postmortem examination confirmed these observations. The sputum raised by all who had lung involvement was of the same character, consisting at first of a heavy, tenacious mucus, later of a lighter, more frothy material. All of it was heavily stained with black particles resembling soot.

The areas of acute emphysema showed the same migratory tendency as those of atelectasis and lobular collapse. Here, too, auscultation showed that the breath sounds were almost absent and râles were rare but the emphysematous areas, which predominated in the upper lobes, were distinguished by their extreme resonance to percussion. Emphysema was also seen roentgenologically and at necropsy. The areas of collapse and of emphysema appeared to have a common origin in obstruction of the bronchioles—complete obstruction causing collapse, and partial obstruction producing emphysema.

Although breath sounds could barely be heard, the patients appeared to be breathing easily and with normal depth. As one might expect in this type of lesion the vital capacity of the lungs of most of the survivors (previously healthy adults) was markedly reduced, frequently to levels of only 800 to 1300 cc. The vital capacities in 19 patients averaged 73 per cent of the theoretical normal on the seventh day after the fire, with extremes of 25 and 120 per cent. Those which were diminished returned to normal only slowly. This diminished vital capacity is probably not unlike that found in cardiac decompensation. The lack of lung elasticity due to burns and edema probably accounted for it as much as the lung collapse and emphysematous areas. It doubtless was a factor in the anoxia experienced by these patients. The vital capacity test represents our best quantitative index of the severity of lung burns, even though it obviously cannot be used in the first few days after an accident.

Three patients, not previously afflicted with the disease, developed typical asthma. This was clearly present on the second day and persisted for over a week, then it disappeared, for the remainder of the hospital stay. They were relieved by steam inhalations and by adrenalin or by aminophylline several times, though the response was not invariable. This type of reaction can be described best by the reports of Cases 5 and 19. Other

patients with less obvious symptoms but with asthma like breathing also obtained relief from the use of aminophylline. The response to these drugs indicated that the asthma was due in part to a muscular constriction of the bronchi which could be relaxed for it appears quite unlikely that the drugs would materially reduce the mucosal edema of the bronchioles. It is very interesting that intense asthmatic breathing could be precipitated by the bronchiolar lesions due to inhalation burns.

The lung complications encountered may be classified into four degrees of severity.

Grade 1 (9 patients.) This group showed minimal abnormal physical signs manifested by rales. There was no significant diminution of vital capacity where it was estimated.

Grade 2 (8 patients.) The second group showed rales and emphysema; there was marked diminution in breath sounds together with roentgenologic evidence of trapping of air. There was slight diminution of vital capacity in all. (This grade is illustrated by Case 36.)

Grade 3 (7 patients.) The third degree of severity added persistent atelectasis, attributed to edema sufficiently marked to obstruct the passage of air into certain areas of the lungs during either phase of respiration. Vital capacities were reduced in varying degrees between the limits of 25 and 83 per cent of the theoretic normal. Cases 5, 6 and 19 who showed special features of interest are described as examples.

Grade 4 (12 patients.) This group included the patients with the most severe degree of injury. Seven died (Case 27 is discussed). Five survived (Case 20 is described).

#### CASE REPORTS

Case 36.—This case illustrates Group 2 of lung complications—rales and emphysema. The patient retained consciousness throughout. On admission, he was cyanotic and there were burns of the mouth and nose. For 48 hours he was dyspneic. There were markedly diminished breath sounds over the right lower lobe and rales at both lung bases. He could phonate only in whispers. The roentgenogram showed a small area of atelectasis on the fourth day and later also evidence of some trapped air. After 12 days, his chest was clear except for occasional rales but he was still raising a half ounce of sputum. His vital capacity which was 58 per cent of normal six days after the fire, had risen to 79 per cent three days later. On discharge after seven weeks in the hospital there was no evidence by physical examination or roentgenogram of residual damage to the lungs.

Case 5.—This case illustrates Group 3 of the degrees of lung complications—rales, emphysema, and persistent atelectasis plus asthma. This man, whose mother has asthma, had never had any previous manifestation of allergy beyond slight exertional dyspnea with chest colds; he had a heavy cold at the time of the fire.

He was admitted unconscious and cyanotic, with burns of the lips, mouth, nose, tongue, and trachea. There were rales in both chests. Twenty-seven hours after admission, his breathing became much more difficult, and was relieved following intra-venous aminophylline. This was interpreted as bronchial spasm superimposed upon bronchial edema. The asthmatic breathing persisted for four days and rales with suppression of breath sounds, for eight days in all. Roentgenograms showed air trapping and atelectasis. His vital capacity rose from 54 per cent of normal on the sixth day to 69 per cent three days later.



This patient was seen one month after the fire, at which time there was complete freedom from symptoms together with a normal roentgenogram

**Case 6**—A female, age 16, well except for previous sinusitis. In the fire she lost consciousness, and awakened in the hospital, nauseated and vomiting. On entrance, râles were heard in the lungs, though the nares, mouth, and pharynx showed evidence of the effect of the heat. She was not otherwise much burned, but was in mild shock, the systolic pressure falling from 120 to 80 mm Hg, which was improved by a plasma transfusion. Within three hours, she developed hoarseness, with moist râles throughout both chests, signs of consolidation in left lower lobe, and showed marked cyanosis, an elevation of respirations to 40 per minute. She was much improved in color and breathing by oxygen, by Boothby mask, after aminophylline had failed to relieve her. However, she became more restless, raised tenacious mucus, and 12 hours after the fire she was put in an oxygen tent because of her poor responsiveness, cyanosis, and increasing respiratory difficulty. She had to breathe oxygen-rich mixtures nearly continuously until the evening of the fifth day.

Prior to the third day, physical signs indicated transient blocking of the lower bronchi, but thereafter there remained a constant block of the lower portion of the lower lobe where constant dullness to percussion and bronchial breath sounds were heard (unique in this series). The rest of the chest examination showed constant and marked diminution of breath sounds with variable and migrant areas of râles, dullness and hyperresonance though collapse of the right lower and emphysema of right upper lobe were usually found. She could exert no force on expiration or cough and could not dislodge the mucus plugs in her bronchi.

She gradually improved until the eleventh day, when it appeared safe to tip her and chest to drain the lung. This had a remarkably good and rapid effect, and though she raised no sputum the lung collapse in both lower lobes disappeared, aeration thereafter was much improved and the diaphragms descended well for the first time. Her vital capacity rose immediately from 0.8 liters (25 per cent theoretic normal) to 1.4 liters (37 per cent of normal), and by the end of the third week this had returned to 3.0 liters, or 90 per cent of normal. Her convalescence was uneventful.

**Case 19**—A male, age 42, with no previous history of asthma or chronic cough was largely a problem of inhalation burns, with minor burns particularly evident on the nose and mouth. On entrance, he had râles in both lung bases followed, six hours after the fire, by signs of blocking of his right lower lobe. Six hours later he had asthmatic breathing and moist râles and expiratory stridor, mostly in right upper lobe. After 29 hours, he became very ill, with intense dyspnea, evidence of obstructed bronchi of right lower lobe, and universal râles, and later, signs of emphysema in right upper lobe. Tracheotomy was considered but not done. Asthmatic breathing from the beginning was relieved by aminophylline. On the third day, he could not phonate audibly and local examination disclosed diffuse swelling and redness throughout the nasopharynx, with chest signs of diffuse emphysema sufficient to depress the diaphragm and obscure the heart. The breathing was typically asthmatic and the clinical picture consistent with asthma of long standing. The patient was very ill, required repeated stays in an oxygen tent, and was frequently relieved by aminophylline and codeine as well as by adrenalin injections. This continued with gradual improvement, with increasing amounts of mucoid sputum (containing 1.8 Gm % protein) and lessening of asthmatic breathing and physical chest signs. The vital capacity was greatly reduced by expiratory push-test (see Schatzki) and after ten days was 1.4 liters, only 34 per cent of the theoretic normal and 74 per cent of normal by the nineteenth day. At the end of a month the lungs still sounded far from normal. There was no dullness to percussion, but breath sounds were very distant and faint over the whole back, most markedly down the right lower lobe. Many coarse bronchial râles were heard over the left lower lobe, particularly after coughing. Vital capacity was 2.6 liters, or 65 per cent

theoretic normal. The patient felt weak but well complained of no asthma or shortness of breath, and only of the persistent cough and sputum.

**Case 27**—A female age 18, was very seriously burned. On admission her color was slightly cherry red, and she was wildly delirious. After an hour she developed respiratory failure she was given artificial respiration followed by oxygen and carbon dioxide an airway was inserted and she was then put into an oxygen tent. One hour later her lungs were wet her systolic blood pressure varied from zero to 120 she was unresponsive, and six hours later she was still unresponsive. Her lungs were full of râles. Given amnophylline, she awakened and coughed and repeated tracheal aspiration produced a 7 cm. bronchial cast streaked with black pigment. Roentgenograms 11 hours after admission, showed partial collapse of the right upper lobe and probably of the middle lobe. There was also acute dilatation of the stomach and esophagus. The next roentgenogram after a four hour interval during which a Levine tube had been used, showed that the dilatation of the stomach had decreased and the esophagus was no longer dilated.

The next morning her breathing was labored, almost Cheyne-Stokes in character and a patch of bronchial breathing was heard at the base of the right lung. An electrocardiogram showed sinus tachycardia, with a rate of 150 the tracing was otherwise within normal limits.

She continued to have a good airway for a day then developed gurgling noises in her trachea. Suction with an intratracheal tube evacuated a very thick mucus but it could not all be removed. The patient became cyanotic and died 62 hours after admission. An autopsy was performed.

**Case 30**—This case represents the most severe grade of lung complications encountered among the survivors. This patient, a female, was one of those who were badly burned, and her dressings made examination of the chest unsatisfactory. She was cyanotic and had râles on admission, but did not begin to cough until about 24 hours later. During the next few hours she sounded like an asthmatic, until she coughed up a plug of pinkish-black material, which suggested a bronchial cast. On the third day following the fire she developed a massive collapse of the right lower lobe. Laryngoscopy showed definite edema of the cords and a tracheotomy was performed. That evening she was unconscious and had a respiratory rate of 38 in her oxygen tent. The lung collapse diminished in the course of about 24 hours, though localized areas of atelectasis remained for 20 days. During the period of reexpansion she produced many brown mucoid plugs.

She improved gradually. The tracheotomy tube was removed after 20 days. Scattered wheezes, clearing after deep breathing were heard for some days more. A roentgenogram in the eighth week after the fire, showed no evidence of any abnormality.

The sequence here was partial bronchial obstruction due to a plug associated with clinical asthma. Subsequent complete plugging produced massive collapse. The patient survived this acute phase and the bronchial edema subsided. As laryngoscopy showed only edema and no third-degree burn of the cords it is improbable that she had a deep burn of the bronchial mucosa. Seven weeks after the fire her lungs were apparently normal, as determined both by physical and roentgenologic examination.

Therapy for severe lung complications of burns is difficult and in a sense inadequate but it is important that it be carried out vigorously and with a clear conception of the underlying pathology. At first the problems are essentially those of getting sufficient oxygen to the lungs of reducing edema of the mucous membranes and of avoiding pulmonary infection. Oxygen in high concentration is the obvious emergency therapy. Patients

receiving morphine should be watched closely. Although intubation and tracheotomy were not highly successful in our cases, we believe that they fulfill a definite function in relieving labored breathing and in facilitating the delivery of oxygen, and should be resorted to in patients with acute cyanosis and in those with severe upper respiratory lesions. Intravenous saline solution is contraindicated since it will increase edema and exudate in the bronchial tree, plasma transfusions will apparently not do this if given in moderate amounts, and obviously they are the essential therapy after burns. It should be remembered that loss of fluid from burned areas of skin and superficial edema can occur with little harm, while similar occurrences within the respiratory tract may be dangerous.

It is, of course, most important to avoid infection. None of the patients in this group developed pneumonia or pulmonary abscess, and this was in all probability due to their isolation on one floor and the precautions taken to avoid cross infections by the continuous use of masks and gowns, and by scrupulous cleanliness. The early use of sulfadiazine was probably also an important factor.

In the later subacute phase, the main problem was to lessen the viscosity of the sputum and to liberate blocked bronchi. A moist atmosphere, produced by means of steam kettles and by liberating steam from the room heating units, gave considerable comfort and improved breathing. Aminophylline appeared helpful, not only in the asthmatics but also in the other dyspneic patients. To loosen secretions, ammonium chloride appeared to be of some value. Gravity drainage during convalescence had a dramatic effect in liberating one collapsed lung (Case 6), and should be used when the patient is well enough to endure it.

#### CONCLUSIONS

Of the numerous lessons to be learned from these lung burns, the following should be emphasized: (1) That covering the mouth with a wet cloth may afford complete protection against pulmonary burns, (2) that in most patients the degree of inhalation burn was by no means ascertainable directly after the fire, and the extreme edema, which occurred later could not be predicted, (3) the resuscitation of patients in acute attacks of edema was difficult and unsatisfactory, and these acute attacks must be watched for with great vigilance, even in patients with minimal surface burns.

The pulmonary complications were bizarre and characterized by extreme variability, with areas of lung collapse and emphysema, which were often quite transient and migratory. As the injury to the bronchioli healed, these signs disappeared and the lungs sounded as though no permanent damage had occurred. Roentgenologic examination confirmed this, but only time will tell whether bronchial scars will constrict and produce bronchiectasis in the future.

# ROENTGENOLOGIC REPORT OF THE PULMONARY LESIONS

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IN the first 12 hours the role of the Roentgenologic Department in regard to the Coconut Grove disaster seemed to be that of an observer. This impression was caused primarily by the surprising absence of fractures among the survivors of the fire. In fact only in two of these was there enough clinical suspicion to warrant any roentgenologic examination and in neither of these was a fracture found.

The first roentgenologic examination of the lungs was made on the morning following the disaster that is 12 hours after the beginning of the fire and approximately 10 to 11 hours after patients had reached the hospital. By this time it had become apparent that the majority of the survivors were suffering primarily from some pulmonary complication the nature of which was not clear. Test films with a portable machine, were taken on a few selected patients. A bizarre appearance of the lung fields was noted in most cases while the lungs appeared normal in others. Clinically unsuspected marked dilatation of the stomach was found in several of these chest films. The results of the trial examinations seemed important enough to justify continuing the studies on a more extensive basis.

The roentgenologic investigation began under severe handicaps. A great proportion of the patients was seriously ill, some were unconscious and most were extensively bandaged. All examinations had to be performed on the ward which had become an Isolation Ward. During the first 24 hours a portable machine (10 m. Amp.) was used for the radiographic work. On the second day following the disaster November 30 a mobile condenser discharge unit (General Electric) with a capacity of  $\frac{1}{4}$  mfd. was set up in one of the sun parlors on the ward and was left there for the duration of the isolation period. The patients were wheeled in their beds to this room and radiograms at six feet distance were thus obtainable, except in the cases of a few very sick patients.

The importance of the films taken at inspiration and expiration was soon realized and all examinations therefore included both phases of respiration. Lateral films were taken only in exceptional cases and were not very successful due to technical difficulties.

Thirty nine cases were admitted to the hospital ward. Four of them were discharged before a roentgenologic examination was made (Cases 3, 21, 24 and 31). The remaining 35 patients had one, and most of them repeated roentgenologic studies of the chest. It showed evidence of pulmonary pathology at some time in 22, whereas the examination of the other 13 was negative at all times.

The roentgenologic appearances were bizarre and changed from patient

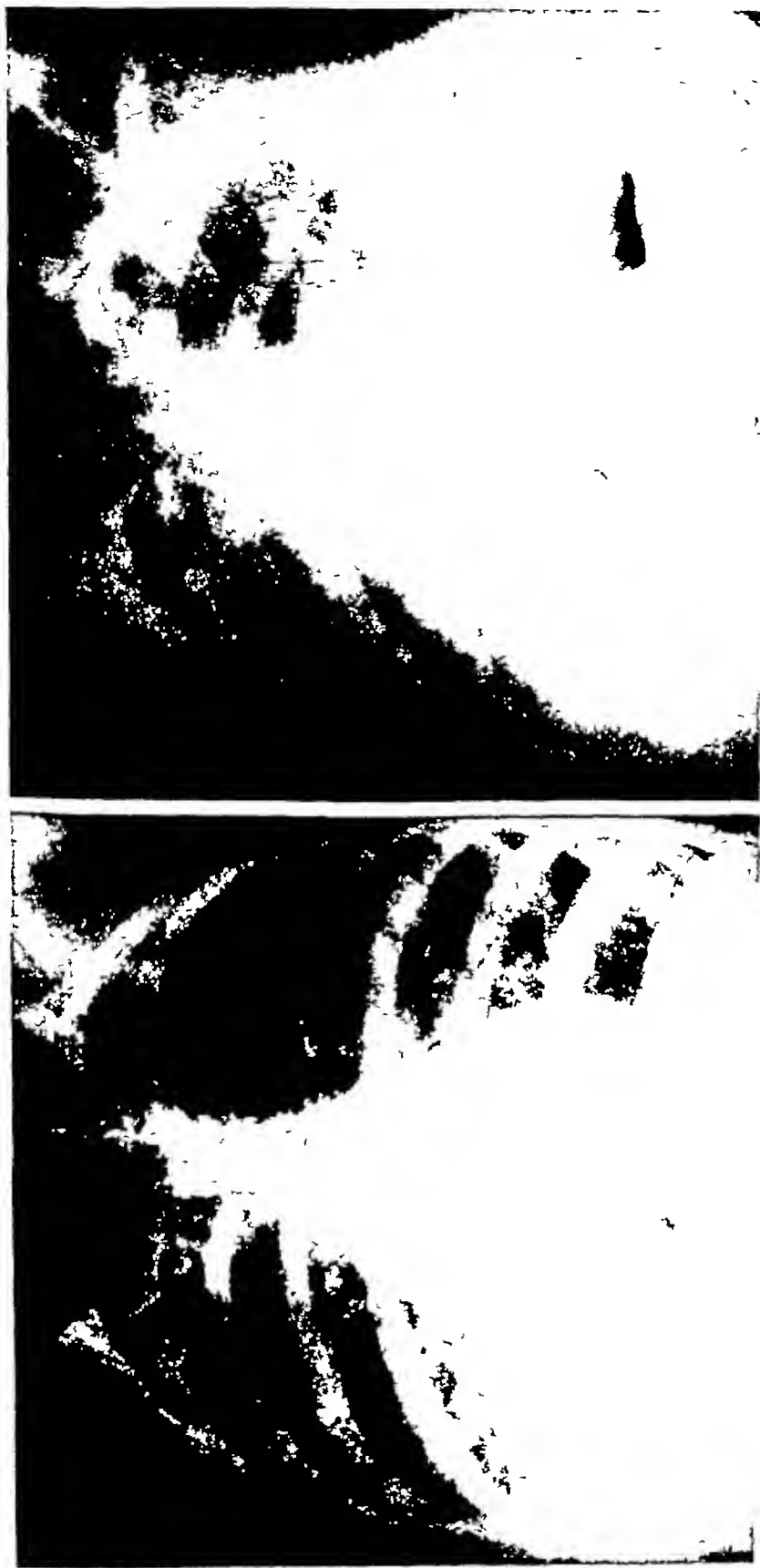


FIG. 8 —Postmortem films taken on two victims of the fire who were dead on arrival at the hospital. The lungs in both cases show extensive pulmonary edema, which was confirmed at autopsy.

## X RAY OF PULMONARY LESIONS

to patient Flame-like areas radiating asymmetrically from one or both hili bands and lines of increased density large homogeneous and small miliary areas of increased density were found as well as areas of increased brightness In general the pictures presented were quite puzzling Some of the lesions were obviously produced by bronchial obstruction while the significance of others was doubtful The further development of the roentgenologic changes as well as the anatomic findings in the few cases which came to autopsy clearly showed that interference with aeration due to complete or partial obstruction of the bronchi and particularly of the smaller bronchi was the cause of most if not all visible pulmonary changes



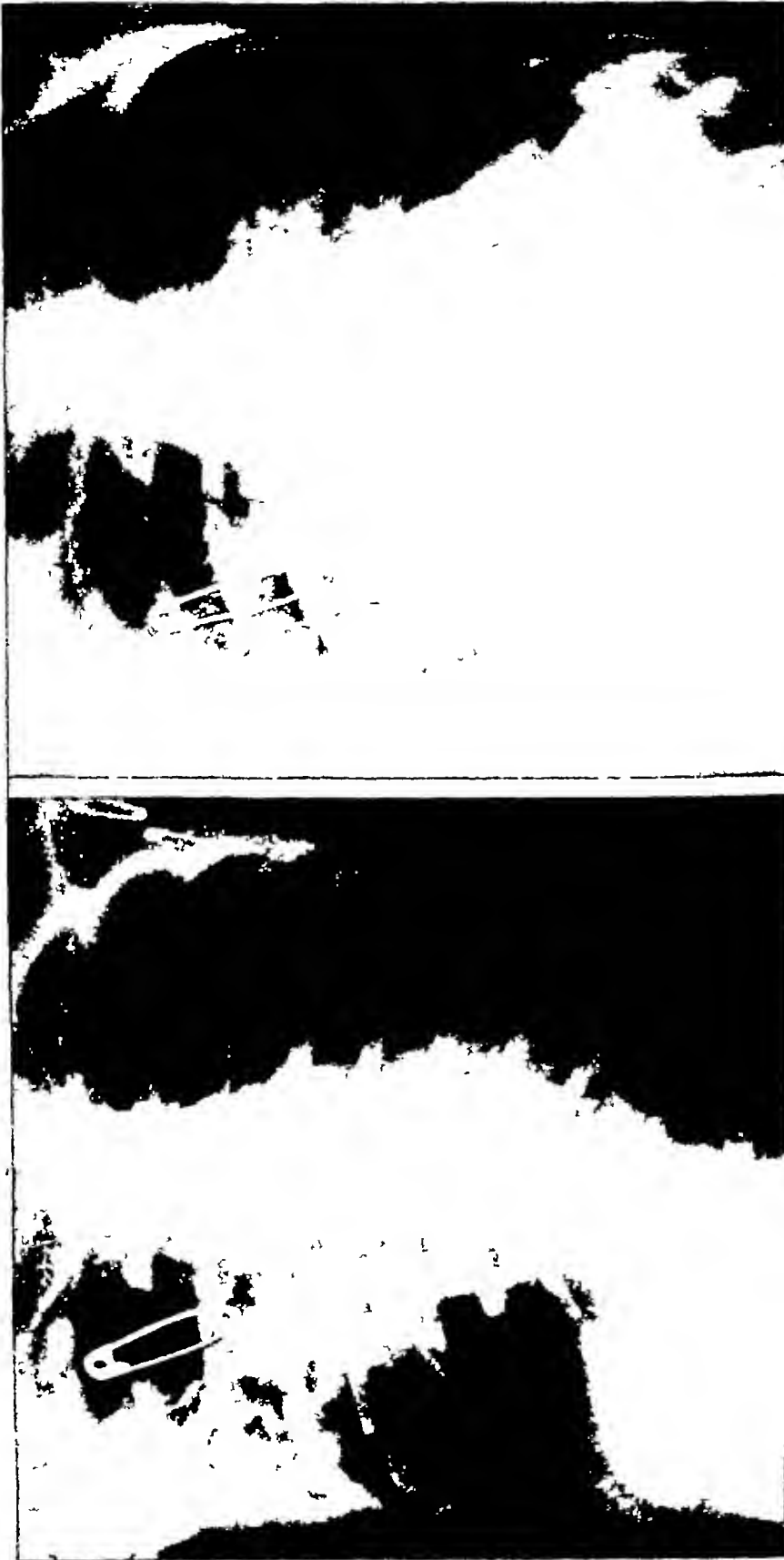
FIG. 9—Case 7 Male, age 29 Second day p.d. Atelectasis in the left lower lobe behind the heart, more clearly visible on the original film. Emphysema of the rest of the left lung The left hilus is decreased in size and displaced downward. Small plate-like foci of atelectasis in the right lower lung field. Patient died 15 hours later The postmortem film showed additional terminal pulmonary edema. Autopsy

### PATIENTS WITH PULMONARY PATHOLOGY BY ROENTGENOGRAM

The findings in the autopsied cases will be described first before analyzing in greater detail the various roentgenologic changes

#### *Roentgenologic Findings in Two Autopsied Cases Dead on Arrival*

Only two of the victims of the disaster dead on arrival at the hospital had roentgenologic examination of the chest Both cases showed extensive, diffuse, poorly defined haziness extending throughout the greater portion of both lung fields and having the characteristic roentgenologic appearance of pulmonary edema (Fig 8) The presence of pulmonary edema was con



A

B

FIG. 10—Case 27 (A) First day, 12 hours p.d. Extensive areas of atelectasis in the right lung field, marked dilatation of the stomach and of the air-filled esophagus, which is visible to the right of the dorsal spine. Trapped air in a localized round area superimposed on the right hilus just below the safety pin. (B) Twenty-four hours later. Increase in the areas of atelectasis on the right side, new areas of atelectasis and emphysema on the left side. The localized area of trapped air is still visible in the right hilus.  
*Clinical Data* Female, age 18. Severe inhalation burns and extensive burns of face, back and extremities. Chest full of rales, rapid respiration, becoming labored on the second day p.d. Death 28 hours following the last film (Fig. 3B). Autopsy.



FIG. 3.—Case 2. Twelve hours p.d. Large area of atelectasis close to the left hilus; small plate like area of atelectasis close to the right hilus.  
Clinical Data. Male, age 39. Severe inhalation burns, extensive burns face, neck, shoulders and hands. Patient died 11 hours after roentgenologic examination. No autopsy.



FIG. 11.—Case 11. Patient died 11 hours p.d. Film taken immediately after death shows mottling of both lung fields, particularly marked on the right side. Note discoloration of the stomach.  
Clinical Data. Male, age 41. Fifty per cent body burns. Legs Coarse, moist rales and prolonged expiration. No autopsy.



firmed by autopsy in both cases. The roentgenologic appearance, as well as the findings at autopsy, was quite different from that seen in patients who survived the first 12 hours, in none of the latter cases was extensive pulmonary edema seen. The blood in the two patients dead on arrival showed a high carbon monoxide saturation—42 and 50 per cent, respectively. Whether this, or another cause, was responsible for the pulmonary edema remains a question, but it is very unlikely that the pulmonary edema in these two cases was similar in origin to the pulmonary changes found at a later date in the survivors.

*Roentgenologic Findings in Three Autopsied Cases which Died in the Hospital*

On only three of the seven patients who died in the hospital were autopsies performed. All three had roentgenologic evidence of pulmonary pathology.

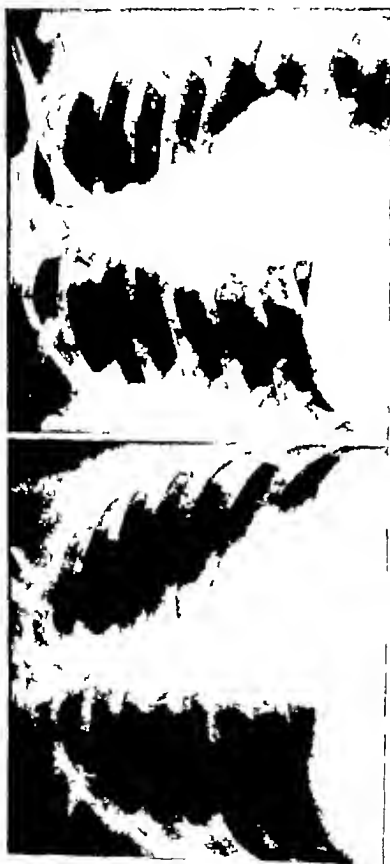
**Case 7.**—In addition to other burns, this patient had burns of the face and severe inhalation burns. A film taken 36 hours after the disaster\* showed emphysema of the left upper lung field, with questionable collapse within the left lower lobe. Another film, taken six hours later, demonstrated more conclusively the atelectasis within the left lower lobe in addition to the emphysema. There were also small plate-like foci of atelectasis in the right lower lung field (Fig 9). The patient died the morning of the third day, 53 hours p d\*. A postmortem film showed pulmonary edema and evidence of trapped air in the left lung. *Autopsy*—Tracheitis, bronchitis and bronchiolitis. Areas of atelectasis with hemorrhage, possibly representing early infarction, the largest area in the left lower lobe, and some pulmonary edema. (See Dr T. B. Mallory's report for details.)

**COMMENT.**—A patient with marked inhalation burns showed early evidence of emphysema and atelectasis, confirmed by autopsy. The changes were adequately explained by severe bronchiolitis. In addition, there was possible evidence of infarction of the collapsed areas, probably secondary in nature. The pulmonary edema was terminal, and in all probability not specifically connected with the underlying pathology.

**Case 25.**—Severe inhalation burns, gurgling and cyanotic. Temperature 103°–104° F. The first film of the chest, taken 12 hours p d, showed localized increased density in the left lower and middle lung fields, which at that time was thought to be pneumonitis or pulmonary edema, but which was later explained as an area of atelectasis. A film taken 29 hours later showed no appreciable change. The patient died 42 hours p d. Postmortem film showed emphysema of the left upper lobe and peculiar well defined mottled areas throughout the right lung and less pronounced on the left side. *Autopsy* showed extreme membranous tracheitis, bronchitis and bronchiolitis, anatomic emphysema, focal areas of atelectasis, and some pulmonary edema. (See Dr Mallory's report for details.)

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\* For convenience, and to avoid repetition, the abbreviation "p d" will be used instead of the words "after the disaster."



A

B

FIG. 12.—Case 6 (See also Figures 14-16 on same case): This case shows the development of extensive, long-standing collapse of both lower lobes. (A) Twelve hours p.d. Small amount of atelectasis in the left lower lobe. (B) Twenty-four hours later. There is now a large area of atelectasis seen through the heart shadow. Note the downward displacement of the left hilum.

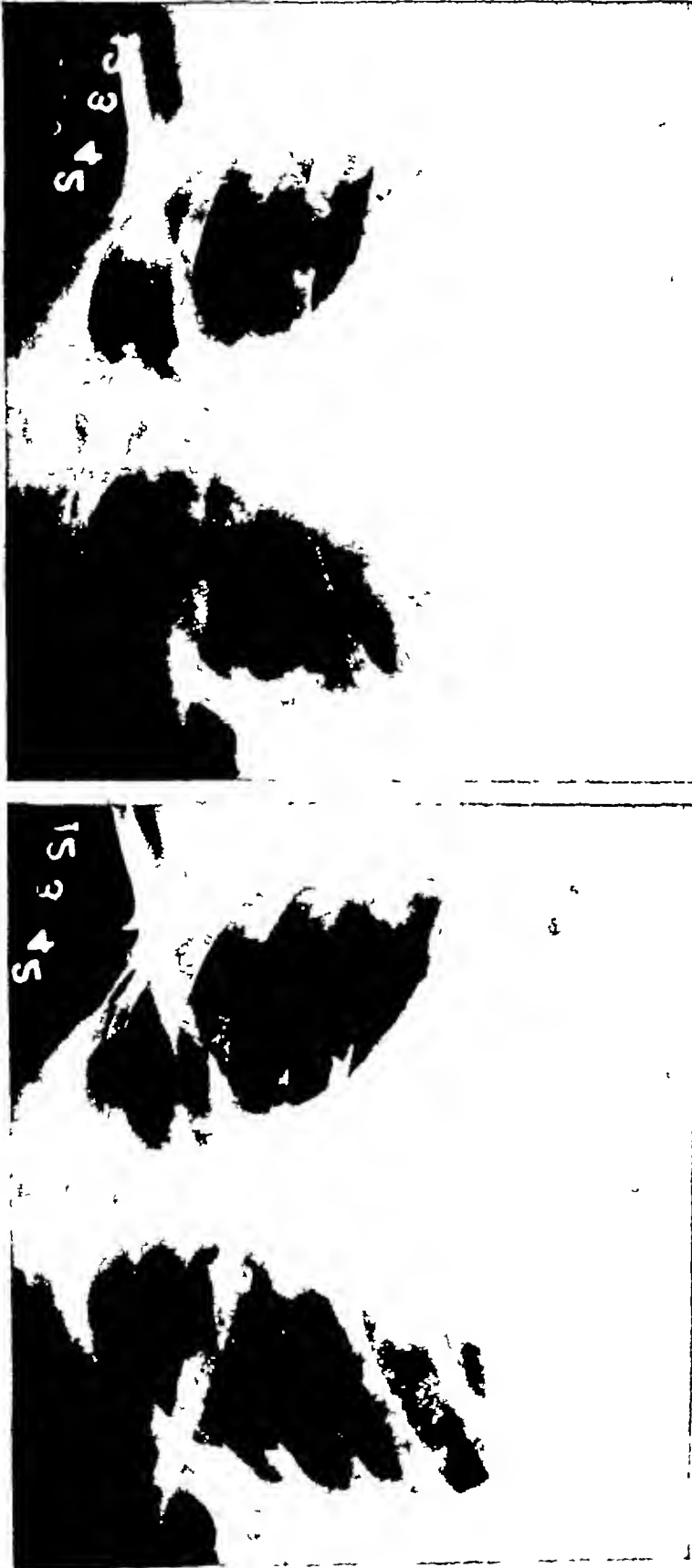


Fig. 14—Case 6 (Continued) Fifth day p d There is now extensive collapse in both lower lobes It became evident on the third day p d, and had not appreciably changed since Note the high fixed diaphragm (A) Inspiration (B) Expiration

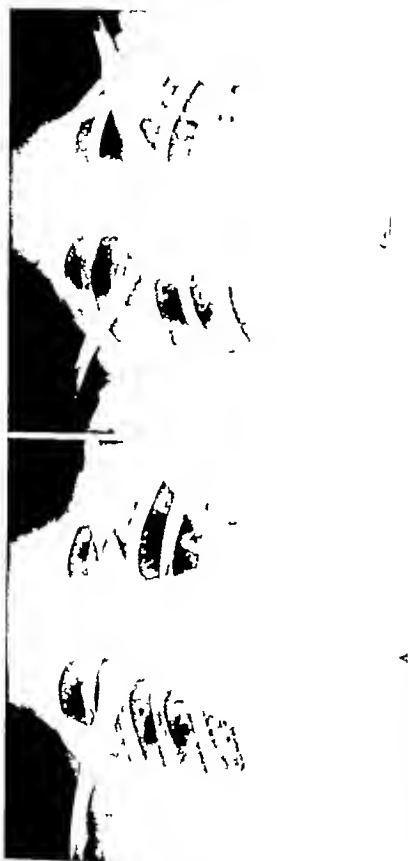


Fig. 15—Case 6 (Cont'd used): Thirtieth day p.d. The collapse of the lower lobes has improved and there is some motion of the diaphragm. (A) Inspiration. (B) Expiration.

COMMENT—Areas of atelectasis and emphysema were present on the first film taken 12 hours p d. The peculiar mottling of the lung seen in this case and in one other fatal case (Case 33) (Fig 11) could be explained either by the demonstration of the exudate-filled small bronchioles or by multiple small areas of atelectasis.

Case 27—Severe inhalation burns, in addition to other burns. Chest full of râles. Films taken 12 hours p d (Fig 10 A) showed extensive plate-like areas of atelectasis in the right upper lung field with elevation of the right hilus. Below this region there was a peculiar flame-like area of increased density. A definite circular 4-cm.-area of increased brightness was superimposed upon the right hilus. The stomach was markedly dilated and air-filled, as was the esophagus. Film taken five hours later showed the stomach and esophagus no longer dilated (the air had been aspirated). The flame-like process in the right midlung field had increased in extent. Films taken 29 hours p d showed disappearance of the atelectasis in the right upper lobe, and slight decrease of the process in the right middle lung field. At this time, there was markedly increased density in the right lower lung field. Films taken 35 hours p d showed no appreciable change on the right side, but there were definite areas of increased density close to the left hilus as well as in the left lower lung field. There were some emphysematous changes on the left side (Fig 10 B). The patient died 63 hours p d. Autopsy showed severe tracheitis, bronchitis, and bronchiolitis, with patchy areas of atelectasis and emphysema in both lungs. Areas of early pneumonia, probably not older than 24 hours. A few small areas of early or partial infarction (See Dr. Mallory's report for details).

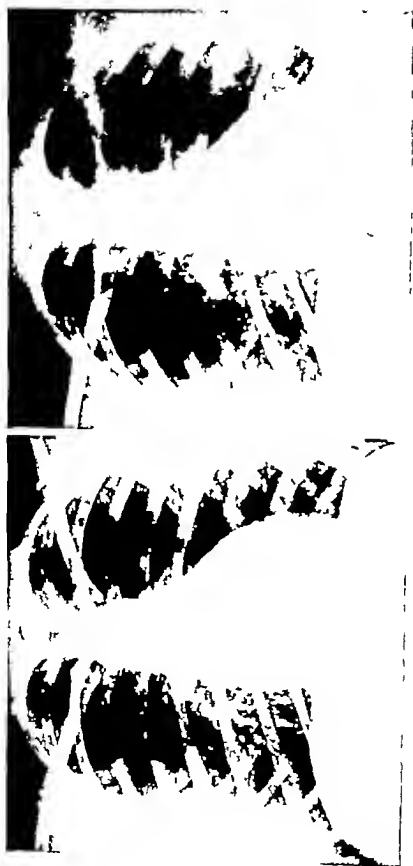
COMMENT—This case shows particularly well the bizarre roentgenologic appearance of some of the atelectatic areas as well as the rapid change in their amount and location. Infarcts may have been responsible for a few of the roentgenologic changes. A perfectly round area of localized emphysema existed in the right middle lung field. It had the appearance of trapped air as sometimes seen in children's pneumonia (pneumatocele).

#### ANALYSIS OF THE ROENTGENOLOGIC FINDINGS

As mentioned before, the roentgenologic findings in the victims of the disaster varied from patient to patient, and often in a given patient from day to day. The resulting pictures were peculiar, the published illustrations giving only an incomplete impression of the variety and combinations. An analysis of the manifold appearances, however, shows that the majority of the changes can be explained by the presence of various degrees and amounts of (1) atelectasis and (2) emphysema, both apparently due to bronchial obstruction. These two types of lesions represent the vast majority of the changes. In addition, there are in a few cases other abnormalities, the nature of which is less clear, which will be described here as (3) areas of milky mottling, and (4) areas of diffuse density without decrease in size of the involved portions of the lung ("drowned" lung). The four types will be discussed in the following paragraphs.

#### *Areas of Atelectasis*

Sudden, massive lobar collapse, comparable to postoperative collapse.



A

B

Fig. 16.—Case 6 (Continued): Fourth week p.d. The collapse of the lower lobes has almost completely disappeared. The left hilus is still slightly lower than normal. The diaphragm shows marked improvement in respiratory motion. (A) Inspiration. (B) Expiration.  
*Clinical Data.* Female, age 6. Marked inhalation burns only minimal skin burns. Early respiratory embarrassment which was quite striking throughout the first few days p.d. Expiratory "push" absent vital capacity markedly diminished. Examination in the 16th week p.d. failed to show any evidence of pathology either roentgenologically or clinically. (For further detail see Dr. Abbott's report.)

was seen in one patient only (Case 20) (Fig 17 B). It was obviously produced by obstruction of a large bronchus. Gradual complete collapse of both lower lobes was seen in another patient (Case 6) (Figs 13, 14, 15 and 16). More commonly, the obstruction was in smaller bronchi and resulted in collapse of the corresponding lobules, usually visible as triangles or bands of increased density (Figs 10 and 23 A). Often the small areas of atelectasis were visible as fine lines only, representing disk-like areas of atelectasis seen edge-on (Fleischner's<sup>5</sup> lines). They ran horizontally, obliquely, and in the lower lobes even vertically through the lung fields (Figs 21 and 22 B). In addition to the autopsy findings, there were several roentgenologic factors which proved the atelectatic character of these lesions. If they were extensive, the noninvolved parts of the lung on the same side showed compensatory emphysema. The diaphragm was commonly elevated on the involved side. Most characteristic were the changes in position and size of the hilar shadows, which were displaced up or down towards the atelectatic lobes, and were smaller on the involved side. In many instances these hilar changes were the first clue towards finding small areas of atelectasis.

A lateral shift of the mediastinum was rarely seen, apparently due to the frequent bilateral pulmonary involvement.

A connection between the roentgenologic findings and bronchial obstruction was demonstrated early by the use of a simple clinical test—the study of the expiratory “push” (forceful expiration with open mouth against the examiner's palm) (Volhard<sup>11</sup>). No stream of air could be felt in patients with marked pulmonary changes, and in others the stream was diminished. The expiratory “push” improved parallel with the roentgenologic clearing of the lungs. This parallelism was later confirmed by more exact studies of the vital capacity.

The majority of the roentgen-positive cases had localized areas of atelectasis at one time or another. Usually several of these were present (Fig 21), at times only one or two. While atelectasis was demonstrable in most of the severely damaged cases on the first day p.d., this pathology was not seen in other cases until the second day p.d., sometimes not before the third and fourth day. In one patient (Case 13), only localized emphysema was demonstrable for a period of ten days, when the first evidence of atelectasis was present in the same region. In some instances, the areas of atelectasis changed from day to day. Usually, however, the same areas of atelectasis remained visible until the process had cleared up. In most cases the pulmonary pathology disappeared within the first two or three weeks, in a few cases it was demonstrable at a later date, up to four or five weeks, and in one case, a small area of atelectasis still existed 17 weeks p.d.

Chart I shows the time of appearance and disappearance of the areas of pathology and also indicates how long after the disaster check-up films were taken.







A

Fourth day p.d. (Continued) Inspiration (A) Inspiration (B) Expiration (B) Expiration There is still marked trapping of air on the right side, although less than previously, minimal areas of atelectasis in the left lower lobe. Many wheezes. On the first day p.d. the patient developed the clinical picture of an asthmatic attack, without preceding history of asthma. Expiratory "push" was absent, markedly diminished vital capacity. The first radiologic evidence of trapped air was seen 12 hours p.d. (Not illustrated here). The emphysema improved gradually but there was still some evidence of trapped air on the right side 18 weeks p.d. (See Dr. Aub's report)

B

*Areas of Emphysema*

Several of the patients showed lobular or lobar areas of emphysema during various stages of their pulmonary complication. They were seen best on films in expiration indicating the presence of trapped air. Areas of atelectasis were usually present simultaneously while in two cases localized emphysema was the only demonstrable pathology (Cases 15 and 39). Persistent lobar emphysema was the outstanding feature in one of the cases and will be described in detail.



FIG. 1.—Case 36: Fifth day p.d. Plate-like foci of atelectasis in both lung fields, a particularly large one in the left lower lobe. This patient showed no roentgenologic evidence of pathology on the first two days p.d. The first appearance of atelectasis, with some emphysema, occurred on the third day. Four weeks p.d. there was fairly extensive atelectasis in the left lower lobe. Nine weeks p.d. the lungs appeared completely normal.

Clinical Data: Male, age 36. Burns of hands, face and cornea. Lungs were clear on the first day p.d. coughing continuously on the second day with bubbles at base. Clinically the lungs cleared up within the first week.

Case 19—Marked inhalation burns with some additional burns. Râles in both bases, with marked wheeze and tracheal rhonchi noted a few hours p.d.

Film taken 12 hours p.d. showed increased brightness in the region of the right lower lobe with some linear areas of atelectasis in this lobe as well as in the left lower lobe and in the bases of both upper lung fields. The right hilus was small. The stomach was dilated and contained a large amount of air. Films taken 30 hours p.d. showed the pulmonary changes more pronounced. Films taken 42 hours p.d. (Fig. 19) revealed the emphysematous changes in the right lung to have increased, and at that time to have involved all of the lobes on the right side with some plate like areas of atelectasis on this side and larger areas of atelectasis on the left side. There was some mottled increased density in the left upper lobe. The trapped air on the right side was

most clearly visible on the film taken in expiration. The right hilus was small and the right diaphragm was low in position. Daily films were taken up to the eleventh day p.d., and from that time on the examinations were spaced at longer intervals. The areas of atelectasis disappeared gradually and had almost completely gone on the fifth day p.d. The trapping of air in the right lung, however, remained clearly demonstrable, particularly on the films taken in expiration (Fig. 20). Eighteen weeks p.d., the right diaphragm was still slightly lower than the left, and there was still definite evidence of air trapping on the right side. The expiratory "push" in this patient had completely disappeared during the first week p.d. It reappeared gradually, corresponding to the increase in vital capacity which initially had been much below normal.

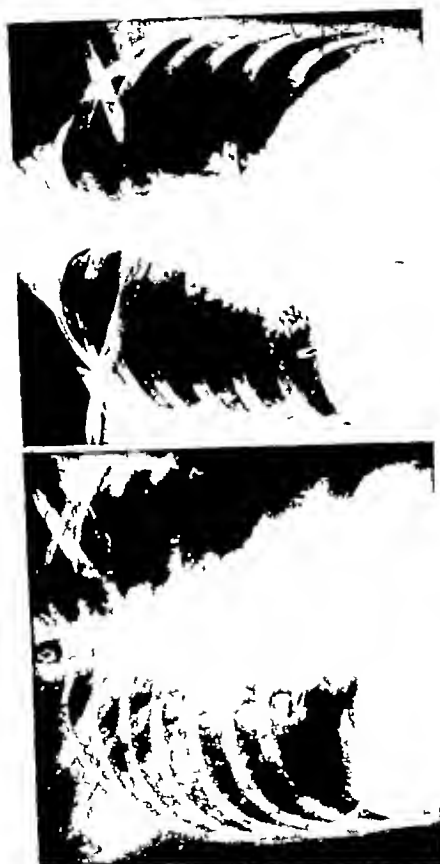
COMMENT—The bronchial obstruction in this patient had produced atelectasis in some areas but had caused mainly ball valve obstruction, particularly of the right lung with resulting obstructive emphysema and a clinical picture of status asthmaticus. Air trapping was still present when the patient was seen last (18 weeks p.d.).

#### *Areas of Miliary Motting*

Peculiar mottling was seen in two of the cases (Cases 25 and 33) (Fig. 11). The changes were particularly marked in Case 33. They consisted of diffuse mottling throughout both lungs, a single lesion measuring from about two to six millimeters in diameter. The lesions were fairly well circumscribed but seemed to be confluent in places. There was also some increase in the linear structures of the lung with several characteristic plate-like foci of atelectasis. The mottling in Case 25, much less marked than in the preceding case, was seen only on the postmortem film, and was present only on the right side. Both patients died. Autopsy was obtained in Case 25 (See above). It did not definitely explain the mottling seen in the roentgenogram. The process simulates somewhat the changes described in bronchiolitis obliterans<sup>1, 9</sup> although in that disease the single lesions are more discreet and smaller. The mottling in our cases is even more like the miliary lesions seen following inhalation of nitrous gases (Nichols,<sup>8</sup> Renander<sup>10</sup>) and of acid fumes (Doub<sup>1</sup>). From the combination of linear shadows and fine mottling in the most outspoken of our cases, it is at least possible that the appearance was produced by multiple fine areas of atelectasis, together with the actual demonstration of plugged small bronchi.

#### *Areas of "Drowned" Lung*

Early in the process three of the survivors (Cases 2, 20 and 32) showed, in addition to the more characteristic areas of atelectasis, an unusual appearance of the lung field which was not seen in any of the other cases. This process was characterized by an homogeneous groundglass appearance covering the lower half of the left lower lobe in Case 20 (Fig. 17 A), the right upper and middle lung field in Case 2 (Fig. 22 A), and the central portions of both lungs in Case 32 (Fig. 23 B). The lesion was noted in Case 20 on the second and third day p.d., and was no longer present on



11

FIG. 32.—Case 3. (A) Twelve hours p.d. Diffuse burn in the right upper lobe ("drowned" lung) ; small areas of atelectasis in the left lower lobe. The pathology in the right upper lobe had disappeared 24 hours later. (B) Eleventh day p.d. Patchy foci of atelectasis in the medial aspect of the right lung and behind the heart on the left side. All these lesions had disappeared by the 7th week, and reexamination in the 17th week p.d. showed normal lungs. (C) Thirtieth day p.d. Extensive inhalation burn in addition to other burns. Great respiratory difficulty. Patient was fibrillating for several hours following admission to the hospital. Later the heart appeared normal. Patient developed clinical and roentgenologic evidence of pulmonary abscess in the 7th week p.d. apparently due to thrombophlebitis of the left leg vein. The lungs however appeared otherwise normal.

the fourth day. It was seen on the first day p.d. in Case 2, and had disappeared on the second day p.d. In Case 32 it did not appear before the second day, decreased on the fourth day, was still somewhat visible on the sixth day, but had disappeared on the seventh day. In contradistinction to large areas of atelectasis there was no evidence of decrease in the size of the involved portions of lung. In Cases 20 and 2 the lesion occurred too early and disappeared too rapidly for pneumonia; whereas it is not possible to rule out pneumonia in Case 32. The appearance was similar to that occasionally seen in asymmetrical pulmonary edema in patients with nephritis or heart failure. Considering the course of the lesions, however, the pathology can best be explained by the accumulation of fluid in the alveoli distal to points of bronchial obstruction. The pathologist Loeschcke<sup>7</sup> describes partial displacement of air by fluid in atelectatic portions of the lung, and the "drowned" lung distal to areas of bronchial obstruction (foreign body, tumor) is a well known clinical occurrence<sup>2, 6</sup>. Fluid was actually found in the alveoli of some of the atelectatic areas in our autopsied cases (See Dr. Mallory's report). The fact that atelectasis occurred later in the originally "drowned" area in Case 20 further supports the validity of the suggested mechanism.

### *Pulmonary Edema*

Pulmonary edema was extensive only in the two victims who were dead on arrival. As discussed before the origin of this pulmonary edema is not known. It may have been caused by carbon monoxide poisoning. In any case, it seems unlikely that it was caused by the same mechanism which produced the pulmonary changes found in the two survivors. One of the patients (Case 7) who died two days p.d. developed pulmonary edema shortly before death, but this terminal pulmonary edema was probably not related to the characteristic pulmonary damage produced by the catastrophe. Aside from this case, pulmonary edema was not seen in any of the survivors, although the question of atypical asymmetrical pulmonary edema arose in three patients. (See paragraph on "Drowned" lung.)

### *Infarcts*

Infarcts were not diagnosed roentgenologically in the first few days following the catastrophe. Two of the autopsied cases (Cases 7 and 27) showed areas in which the alveoli were slightly decreased in size and contained blood but no air. The cell walls were preserved. These lesions were thought to be areas of early infarction, possibly superimposed on areas of atelectasis. (See Dr. Mallory's report.) It is possible that changes which roentgenologically were interpreted as plate-like areas of atelectasis in some of the survivors actually represented small infarcts. It is unlikely, however, that many infarcts were misinterpreted in this manner, inasmuch as the roentgenologic evidence of the lesion commonly changed back-and-forth from day to day, which would not occur in infarcts. Furthermore,

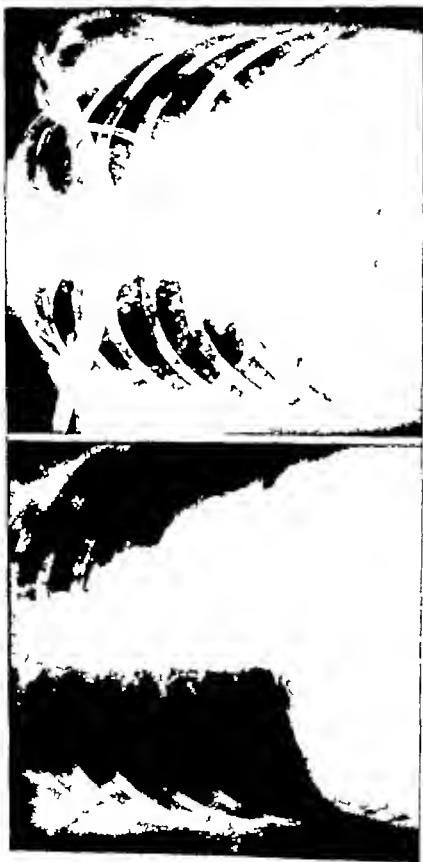


FIG. 23.—Case 22: (A) Twelve hours p.d. Extensive areas of atelectasis behind the heart on the left side as well as in a patchy manner in both upper lung fields. The next day the atelectasis had improved. There were beginning changes in both middle lung fields, more marked on the 3rd day (B). Third day p.d. Extensive pathology in both middle lung fields, the nature of which is not definitely established—drowned? lungs; atelectasis or pneumonia. This process disappeared by the 7th day p.d. but small areas of atelectasis were still visible in the 4th week. The lungs appeared normal in the 7th week p.d. (Fig. 24)

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B

A

FIG. 23.—*Case 12.* (A) Twelve hours p.d. Extensive areas of atelectasis behind the heart on the left side as well as in a patchy manner in both upper lung fields. The next day the atelectasis had improved. There were beginning changes in both middle lung fields, more marked on the 3rd day (B). Third day p.d. Extensive pathology in both middle lung fields, the nature of which I not definitely established—drowned—lungs; atelectasis; or pneumonia. The process disappeared by the 7th day p.d. but small areas of atelectasis were still visible in the 4th week. The lungs appeared normal in the 7th week p.d. (Fig. 24)



all the lesions disappeared without leaving any trace of a scar with the exception of one patient (Case 13) Areas of incomplete infarction may clear up without visible scar,<sup>4</sup> but it is not probable that so few scars would result if an appreciable number of infarcts had been present among the survivors Infarcts did occur, and were seen roentgenologically in some of the severely burned cases several weeks following the disaster, obviously connected with thrombophlebitis

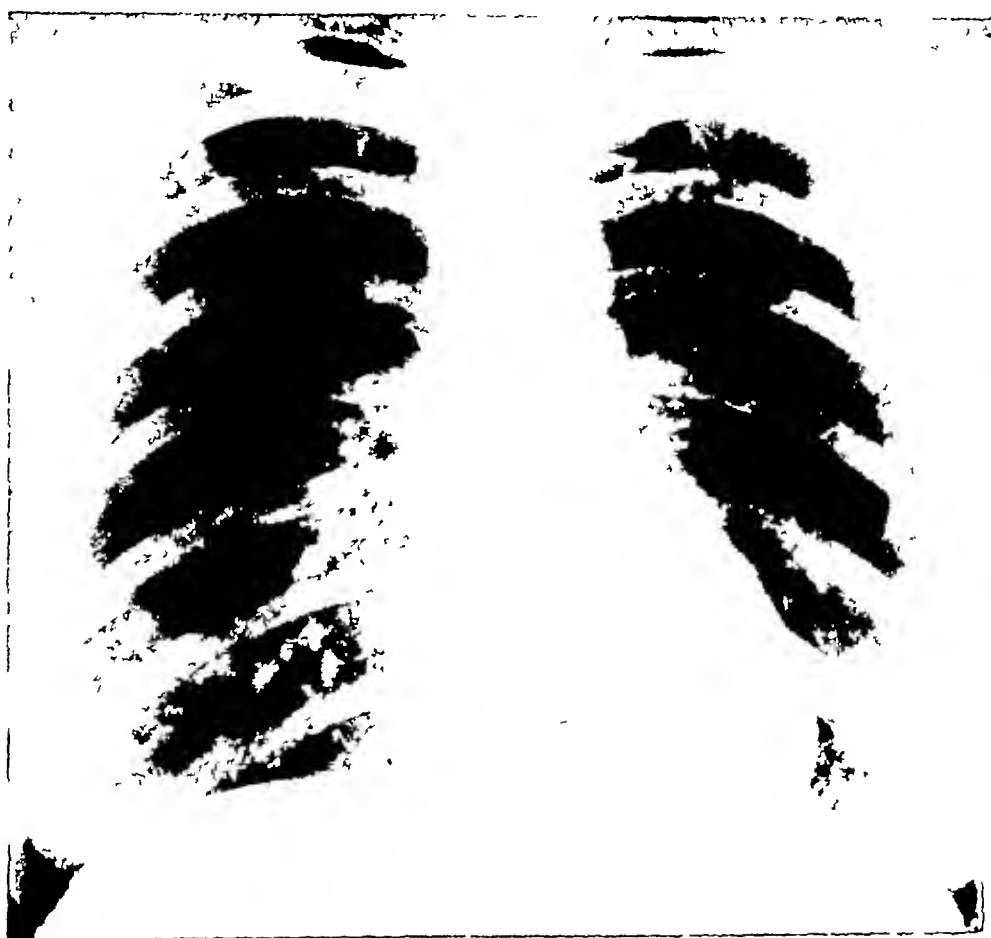


FIG 24—Case 32 (Continued) Normal lung in the 7th week p d  
 Clinical Data Female, age 35 Burns of lips, nose and tongue, moderate burns of forehead and hands Unconscious, severe cerebral complications (See Dr Cobb's report for further details)

### *Acute Dilatation of the Stomach*

Marked dilatation of the stomach was seen on the chest films in five cases (Cases 2, 19, 27, 33 and 34) In Case 27, the esophagus was also markedly dilated and air-filled (Fig 10 A) Three of these patients died It is possible that the dilatation was a sign of general shock although the exact relationship remains obscure

### *Follow-up of the Pulmonary Lesions*

Most of the patients were examined repeatedly, regardless of whether or not the lungs appeared normal roentgenologically These follow-up exam-

inations were continued until finally the roentgenographic appearance of the lungs was normal in all but two cases. One patient showed evidence of persistent air trapping at the end of 18 weeks (Case 19) while another still had a linear area of atelectasis in the 17th week (Case 13).

#### PATIENTS SHOWING NO ROENTGENOLOGIC PULMONARY CHANGES

Thirteen patients admitted to the ward showed no roentgenologic pulmonary changes. One of them (Case 12) died 27 hours p.d. with clinical signs of respiratory embarrassment. These signs developed however some hours after the negative film had been obtained.

All but two of the surviving roentgenologically negative patients presented rales on physical examination on one or several occasions as evidence of some bronchial involvement. In spite of this the roentgenologic examination of the chest failed to show any evidence of pathology. It is likely that the bronchial lesions in these cases were of a minor degree and had not led to interference with the aeration of the lungs.

#### DISCUSSION

This report shows that the abnormal roentgenologic appearance of the lungs in the survivors of the Cocoanut Grove disaster was caused primarily by damage to the bronchi and bronchioles. The roentgenologic changes may be characterized as a classical example of diffuse damage to these parts of the upper respiratory tract. Similar roentgenologic appearances may result from many other causes of widespread bronchial injury and in the recognition of this fact lies the importance of this experience in a broader sense. In infants with bronchiolitis and peribronchiolitis the roentgenograms show areas of atelectasis and emphysema like those described in this report. Damage to bronchioles scattered throughout the lungs from various gases or from inhalation of food or water may produce similar pathology and similar roentgenologic findings. During war time such trauma is likely to be a frequent occurrence and early recognition of the mechanism involved will be of value in the determination of the correct treatment.

#### SUMMARY

1 Thirty five of the survivors of the Cocoanut Grove disaster were examined roentgenologically following their admittance to the hospital. Pulmonary pathology was found at some time in 22 patients whereas the examination of the other 13 was negative at all times.

2 The roentgenologic appearance of the lungs was bizarre and varied from patient to patient. The majority of the lesions however could be explained by areas of atelectasis and emphysema both apparently due to bronchial occlusion, particularly of the smaller branches.

Atypical lesions (miliary nodules and areas of "drowned lung") were seen in a few cases.

Diffuse pulmonary edema was found in two victims who were dead on arrival.

Infarcts were not recognized roentgenologically in the first period following the fire, although some may have existed

3 Acute dilatation of the stomach was found in five patients, and of the esophagus in one patient

4 The follow-up of the pulmonary lesions is described The lesions of all the survivors finally disappeared except in two cases

5 The experience gained from this disaster is of value as it applies to the recognition of the roentgenologic appearance of lungs in cases with damage to the bronchi and bronchioles from other causes

# REFERENCES

- <sup>1</sup> Assman, H *Klinische Röntgendiagnostik der inneren Erkrankungen*, Leipzig, 1929
- <sup>2</sup> Bowen, D R *Acute Massive Collapse of the Lung* *Am J Roentgen*, **21**, 101-141, 1929
- <sup>3</sup> Doub, H P *Pulmonary Changes from Inhalation of Noxious Gases* *Radiology*, **21**, 105-113, 1933
- <sup>4</sup> Hampton, A O, and Castleman, B *Correlation of Postmortem Chest Teleroentgenograms with Autopsy Findings With Special Reference to Pulmonary Embolism and Infarction* *Am J Roentgen*, **43**, 305-326, 1940
- <sup>5</sup> Fleischner, F, Hampton, A O, and Castleman, B *Linear Shadows in the Lung* *Am J Roentgen*, **46**, 610-618, 1941
- <sup>6</sup> Jackson, C *The Drowning of the Patient in his Own Secretion* (editorial) *Laryngoscope*, **21**, 1183-1185, 1911
- <sup>7</sup> Loeschke, H *Störungen des Luftgehalts* (in Henke-Lubarsch *Handbuch d speziellen pathologischen Anatomie und Histologie*, Vol 3, 1st part, Berlin, 1928
- <sup>8</sup> Nichols, B H *The Clinical Effects of the Inhalation of Nitrogen Dioxide*. *Am J Roentgen*, **23**, 516-520, 1930
- <sup>9</sup> Rabin, C B - *Radiology of the Chest* (in Nelson's *Diagnostic Roentgenology*, edited by R Golden, Vol I, p 196-Z, 1941
- <sup>10</sup> Renander, A *Röntgenologisch beobachtete reversible Veränderungen bei nitrosen Gasschaden an den Lungen* *Acta Rad*, **17**, 152-159, 1936
- <sup>11</sup> Volhard, F *Personal communication*.

## **PATHOLOGY WITH SPECIAL REFERENCE TO THE PULMONARY LESIONS**

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SIX POSTMORTEM EXAMINATIONS were performed either at the Northern Mortuary of Suffolk County or in the Pathology Laboratory at the Massachusetts General Hospital. Although these represent a very small percentage of the victims of the holocaust, the findings parallel so closely in most respects the results of some 16 other autopsies performed elsewhere in the city that they may be considered fairly representative. Material for histologic examination from five other cases was also made available to us but provided so little additional information that its formal presentation seems unnecessary. The six cases examined by us personally fell into two groups. Three cases which were dead on arrival and three which died after varying but comparatively brief (40-62 hours) periods of treatment upon the wards.

It was apparent from inspection of the bodies which were delivered to the hospital and to the mortuary that only a small proportion of the victims had suffered extensive enough surface burns to account for death and that a search for contributory factors was in order. The most obvious of these was carbon monoxide poisoning which was apparent clinically and was confirmed at autopsy by the finding of 42 per cent and 50 per cent saturation of the hemoglobin in samples obtained from the right heart in two cases dead on arrival. Although significant as a contributing factor this degree of monoxide saturation could not in itself be considered lethal. In one case a sample of gas collected by suction from the respiratory tract showed on chemical examination traces of oxides of nitrogen with a test sensitive to 50 parts per million. Since a second sample was negative the results must be regarded as equivocal. Beyond this point postmortem studies were entirely in accord with early clinical impressions in emphasizing the great importance of the involvement of the respiratory tract. The bulk of this report will therefore, be devoted to the findings in the upper respiratory tract and in the lungs.

### **CASES DEAD ON ARRIVAL**

The three cases which were dead on arrival presented lesions so similar that only one needs to be recorded. An abbreviated protocol follows.

*Protocol.*—The body is that of a well-developed and well-nourished young woman, estimated to be about age 25. It shows severe second and third degree burns of the head, face, neck, both arms, the chest above the breasts and the entire back to the level of the sacrum, including essentially all skin areas which would be exposed by a conventional evening gown cut relatively low in front and to the gluteal fold behind. Scattered first and second degree burns with extensive vesiculation are present on the legs, particularly the medial surfaces above and below the knees. The entire face and the neck have an edematous, bloated appearance.

With the initial incisions it is apparent that the muscles and most of the internal organs are a brilliant cherry-red in color. This is not apparent in the blood that escapes from severed vessels or in the chambers of the heart which appears dark red. A sample of this blood, however, submitted for chemical analysis showed 42 per cent saturation with carbon monoxide.

The trachea and larger bronchi contain considerable amounts of partially digested food similar in character to that which is present in the stomach. The underlying mucosa is diffusely reddened and shows numerous pin-point hemorrhages which are most frequent in the upper trachea and decrease as one descends the bronchial tree but scattered foci can still be found in bronchi 3 mm in diameter. No exudate is present and no froth.

The lungs weigh 1230 Gm together. They are bright pink, heavy, voluminous, and do not collapse (colored section Fig 1). Crepitation is uniformly diminished but nowhere entirely absent. Fragments of the tissue float in water. The pleura is smooth, glistening and transparent, the underlying parenchyma brilliant pink in color. Several subpleural hemorrhages 5 to 10 mm in diameter are found over the lower lobes. On section, all lobes present uniform, congested but very bright red surfaces. On slight pressure considerable quantities of blood-stained fluid ooze from the freshly cut surfaces but this is not frothy. The pulmonary arteries and veins are normal.

The heart, liver, kidneys, spleen and internal genitalia are normal except for cherry-red discoloration of all blood containing areas. The gastro-intestinal tract is normal except for congestion and petechial hemorrhage throughout the duodenum. The brain shows only an unusual pinkish tinge to the grey matter.

*Microscopic Examination.* The trachea and primary bronchi show essentially similar changes (Fig 25). All vessels internal to the cartilaginous ring are maximally dilated. The areolar tissue of the mucosa is markedly edematous and at intervals of 2 to 3 mm shows focal hemorrhages 0.5 to 1.0 mm in diameter. Almost no leukocytes are found outside of vascular lumina. The basement membrane is normal and the majority of the epithelial cells have desquamated. No ciliated or goblet cells persist but a single layer of small cells with scanty undifferentiated cytoplasm adheres to the basement membrane in many places. No coagulation necrosis or eschar formation can be made out.

Several sections from various portions of the lungs present essentially similar pictures. The great majority of the alveoli are of normal or slightly subnormal size and are filled with precipitated eosinophilic material, partly granular and partly homogeneous. In the latter instance it occasionally reaches almost the density of thyroid colloid (Fig 26). In some alveoli considerable numbers of red blood cells are present and these are almost invariably found in association with the homogeneous rather than the granular precipitate. Scattered between these edematous alveoli are bubbles of air ranging from two to three times the size of the alveoli. These are invariably localized in dilated atria and respiratory bronchioles and do not represent emphysematous alveoli (Fig 27). There is, thus, very little intermingling of air and fluid. The capillaries of the alveolar walls and all small blood vessels throughout the lungs are congested. The interlobular septa are widened and edematous, and the lymphatics here and in the perivascular tissues are frequently dilated and contain granular precipitate. The small bronchi show desquamation of epithelium and no necrosis or inflammatory reaction. Nowhere in the sections is there leukocytic infiltration. Several bronchioles and a few alveoli contain aspirated vegetable cells.

Microscopic examination of sections of other organs show no significant variations from normal. The liver cells present the granular, dispersed cytoplasm and prominent cell membranes characteristically seen in cases of sudden death in the glycogen-storage phase of metabolic activity.

The major parenchymal lesion in this case, as well as in the others of



FIG. 25.—Trachea from case dead on arrival. The epithelium is desquamated, the mucosa edematous without leukocytic infiltration. A focus of hemorrhage is present in the superficial portion. The mucosal blood vessels are maximally dilated.

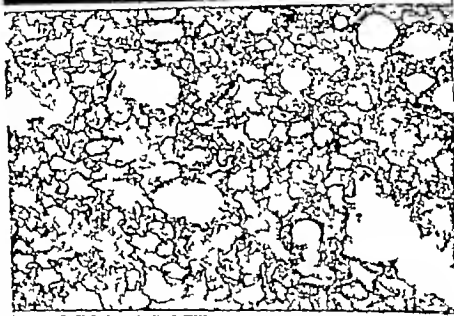


FIG. 26.—Lung of the same case demonstrating massive pulmonary edema with bubbles of trapped air.

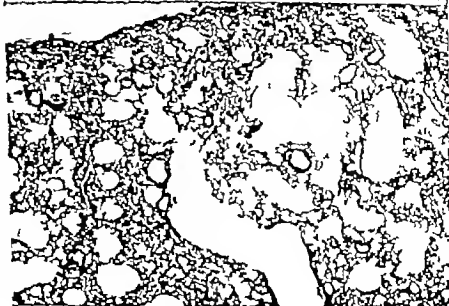


FIG. 27.—Another area demonstrating that the trapped air lies predominantly in dilated respiratory bronchioles and alveoli rather than emphysematous alveoli.

the group dead on arrival, was the diffuse pulmonary edema. A slightly unusual feature was the absence of froth in the trachea and bronchi as well as in the fluid which oozed from the cut surfaces. This appeared to be explained by the fairly sharp limitation of the edema to the alveoli themselves. Such air as was present was found in atria and respiratory bronchioles. There was, consequently, comparatively little actual admixture of air and fluid. A high protein content of the edema fluid was evidenced by the homogeneous colloid-like precipitation in the alveolar lumina, and the presence of red blood cells confirmed the existence of significant capillary damage.

These cases were too long postmortem (36 hours) to attribute safely significance to the desquamation of the tracheal and bronchial epithelium. The absence of any eschar formation and the intact basement membrane, however, make it doubtful, in these cases at least, that flames could have been inhaled far into the respiratory passages. That some physically or chemically irritating agent was so inhaled is indicated by the vascular dilatation, the marked edema of the tracheal and bronchial mucosae and the presence of innumerable pin-point hemorrhages. It seems probable that the pulmonary edema was of similar pathogenesis. The aspiration of vomitus was not responsible since similar changes were found in other cases in which no evidence of such aspiration was present.

#### CASES TREATED IN THE HOSPITAL

Three cases came to autopsy from the group which were admitted to the ward. Case 25 died 40 hours, Case 7, 52 hours, and Case 27, 62 hours after admission. They will be presented in that order.

**Case 25**—The body is that of a powerfully built, large man, age 46. The hair has been extensively singed but the scalp is free from discoloration or vesiculation. There are first, second and third degree burns covering the entire face and upper neck, the areas of third degree burn being comparatively small and localized about the mouth, nose, chin and ears. The mucous membrane of the lips is blistered. The hairs of the nostril are singed, and greyish adherent exudate is visible in the nostrils and overlying the inferior turbinates. The entire face is swollen and edematous, especially the periorbital tissues. The conjunctivae are congested but the sclerae are intact. First, second and third degree burns are likewise found on both hands, particularly on the dorsal surfaces, where they extend up to the cuff line.

**Respiratory Tract** The larynx when viewed from above appears completely occluded by edematous swelling of the mucous membranes, particularly those overlying the false cords and the aryepiglottidean folds. Fibrinopurulent exudate overlies the former. The surfaces are glazed and whitish in color but there is no ulceration or membrane formation (color section Fig 3). When the larynx is viewed from below (color section Fig 4), in contrast, a greenish-black membrane, to which black fragments of charred material are adherent, extends from the inferior surface of the true cords downward, encircling the trachea for a distance of 2.5 cm. The membrane can be stripped from the mucosa only with difficulty and leaves a raw, hemorrhagic surface. The mucous membrane of the remainder of the trachea is intensely red with scattered petechial hemorrhages. Foci of ulceration and membranous exudation are absent for a distance of 4 cm below the zone described above, then begin again as scattered foci 2 to 10 mm in diameter in the middle and lower thirds of the trachea, and become almost confluent again in the primary bronchi.

## PATHOLOGY OF THE RESPIRATORY TRACT

The lungs are extremely voluminous and fail to collapse on removal of the sternum. They are heavy of diffusely increased consistency and are subcrepitant throughout. Along the margins of all lobes, however is a zone 2 cm. in width which is lighter in color and more normal in consistency than the central parts of the lung (color section Fig. 2). Air is evidently trapped in this peripheral zone since it fails to collapse even after removal from the thorax. Several emphysematous blebs are evident at each apex.

The left lung is sectioned at once, the right one saved for lipiodol injection, roentgenologic examination, and fixation before sectioning. On opening the bronchial tree all bronchi down to 3 mm. in diameter show intensely red, hemorrhagic mucosae overlaid by a greenish fibrinous membrane (color section Fig. 6). This is in places rather firmly adherent, in other areas strips readily and in still other places has spontaneously desquamated into the lumen to form occlusive bronchial casts. The lung parenchyma in the central two-thirds of each lobe is deep red in color and moderate amounts of pinkish fluid can be expressed by gentle pressure. A zone averaging 2 to 3 cm. at the periphery of the upper and middle lobes and 1.5 cm. in the lower lobe is paler and comparatively well aerated. Section of the left lung fixed by instillation of formalin into the bronchial tree, shows similar hemorrhagic, membranous bronchitis throughout the major radicles of the bronchial tree. In addition, it shows extensive anatomic emphysema of the upper anterior half of the upper lobe. Through this area the alveoli average 2 to 3 mm. in diameter and numerous bullae 5 to 10 mm. in diameter are present. Minor focal spots of emphysema 2 to 3 mm. in diameter associated with dense anthracotic deposits are scattered throughout the remainder of the upper lobe and also are present in the lower lobe. Slight atheromatous deposits are present in the major pulmonary arteries. The remainder of the autopsy shows moderate cardiac hypertrophy without valvular lesions, slight coronary sclerosis without narrowing of the lumina, moderate atherosclerosis of the aorta, a large, deeply congested but evidently fatty liver, congested kidneys from which the capsules strip with slight difficulty leaving slightly granular surfaces, a fatty pancreas and a right inguinal hernia.

*Microscopic Examination Larynx Trachea and Primary Bronchi:* These show similar pictures varying however from one area to another in the intensity of the reaction. All sections show complete desquamation of the epithelium except in pockets about the mouths of the gland ducts. The basement membrane is in most areas intact. In one section it and the underlying 30 to 40 microns of tissue are necrotic. In another section, evidently from near the larynx, epithelium and basement membrane are missing and have been replaced by a diphtheritic membrane consisting of sheets of parallel fibrinous lamellae with polymorphonuclear leukocytes between the lamellae. In all sections the small vessels are maximally engorged, and there is edema and polymorphonuclear infiltration of the submucosa. In many sections there is likewise evidence of a chronic inflammatory process shown most clearly by foci of fairly dense lymphocytic infiltration in the stroma of the mucous glands.

*Lower Bronchial Tree* The findings vary considerably in different bronchi and bronchioles apparently independently of their size. None are normal, since vascular engorgement and polymorphonuclear infiltration are invariable. In some bronchi the epithelium has partially or completely desquamated and masses of detached but fairly normal looking ciliated and goblet cells are found in the lumina intermixed with mucus and polymorphonuclear leukocytes. In other bronchi the epithelium is still *in situ* on the basement membrane. In these cases the cells may stain normally but show a tendency to separation by edema fluid and leukocytic infiltration. In other places the epithelial cells are recognizable by their elongated configuration or by their location but are completely acrophilic and their nuclei have partially or completely lost the power of retaining the stain. These necrotic epithelial cells are found singly and in clumps in the bronchial lumina where they are combined with leukocytes mucus



## MALLORY AND BRICKLEY

and fibrin to form occlusive bronchial plugs (Fig 28) In one bronchus the mechanism of detachment of the necrotic epithelial cells is clearly shown by the presence of vesicles filled with clear fluid and a few leukocytes between the necrotic cells and the basement membrane Only rarely is there evidence that the basement membrane itself has been destroyed In these areas a typical diphtheritic membrane similar to that noted in the upper trachea is present In many bronchi evidence of a preexisting chronic inflammatory process is present in the form of lymphocytic infiltration of the mucosa and mucous glands

*Lungs* The pulmonary parenchyma proper shows an alternation of partially collapsed air sacs which often contain precipitated edema fluid and overdistended air passages containing trapped bubbles of air The majority of the air-filled structures can be identified as respiratory bronchioles and atria rather than alveoli The presence, however, of true anatomic emphysema, bordered by foci of fibrosis and either anthracosis or lymphocytic infiltration makes the distinction between anatomic and physiologic emphysema very difficult Both are certainly present but their relative proportions cannot be judged with accuracy

The edema fluid in the alveoli is demonstrated in part by granular, in part by homogeneous precipitate It is assumed that the latter indicates a higher protein content There is minimal extravasation of erythrocytes An occasional lobule shows frank pneumonic exudate with many polymorphonuclears, some monocytes and but little fibrin In these areas colonies of micrococci are present and a few cocci can be identified in the cytoplasm of the polymorphonuclears

*Liver* Occasional cells are coarsely and nearly all cells finely vacuolated with fat The space between the liver cords and the sinusoidal endothelium is almost uniformly widened, a finding usually considered indicative of hepatic edema

*Pancreas* Negative except for the presence of considerable amounts of adipose tissue

*Kidney* The intima of the intermediate-sized and larger arteries is thickened with considerable reduplication of the elastic lamellae Small foci of atrophic tubules and an occasional sclerosed glomerulus are seen A few tubules contain bluish-staining hyaline casts

On opening the head, very marked edema of the deep areolar tissues of the scalp is noted The brain is normal except for congestion of the meningeal vessels and edema of the arachnoid

In summary, a forty-six-year-old man, of powerful physique, showed evidence of long-standing chronic bronchitis, moderately severe pulmonary emphysema, slight pulmonary and moderate systemic arteriosclerosis, fat infiltration of the liver and pancreas, slight hypertrophy of the heart and mild nephrosclerosis Upon these chronic lesions were superimposed severe but not extensive cutaneous burns of the head, neck and hands The outstanding lesions were a necrotizing membranous inflammatory process in the upper respiratory tract which produced almost complete laryngeal stenosis and a similar process in the intrapulmonary bronchial tree resulting in diffuse bronchostenosis Though tracheotomy would have relieved the laryngeal stenosis, the degree of bronchial obstruction was so great that no air-way could have been established The pulmonary parenchyma, already handicapped by extensive pulmonary emphysema, was drowned by massive pulmonary edema Only at the periphery of the lung were there narrow aerated zones in which air had evidently been trapped by the diffuse bronchostenosis Gross atelectasis was absent

## PATHOLOGY OF THE RESPIRATORY TRACT

**Case 7**—The positive findings of the autopsy are as follows

The body is that of a powerfully built, well nourished male age 29. Almost the entire face, the scalp and the posterior portion of the neck are covered by first and second degree burns. These are particularly severe and reach third degree in the region of the alae nasi and the margins of the ears. The posterior surface of both hands and wrists running up 3 cm. above the ulnar styloid show third degree burns. Burns are also present on both ankles just above the shoe level. There is a recent tracheotomy incision 5 cm. in length the central portion of which is gaping and from which the tracheotomy tube has been removed. Hematomata are present in both antecubital fossae as well as the scars of several needle punctures overlying the veins. The subcutaneous fat is abundant. The muscles are well developed normal in color and consistency.

**Stomach** Contains approximately 300 cc of greenish brown fluid. The mucosa shows innumerable red puncta varying from 0.5 to 2 mm. in size. These are most numerous at the cardiac end and disappear as the pyloric antrum is reached. No true erosions are found.

**Duodenum** There is moderate congestion and numerous scattered minute petechiae in the mucosa of the second and third portion. The remainder of the intestinal tract is negative.

**Larynx and Trachea** (color section Fig 5) All mucous membranes in the larynx are markedly congested and swollen, producing almost total occlusion at the level of the true cord. Over the arytenoid cartilages the mucous membrane is 3 to 4 mm. thick. Lightly adherent to the mucous surfaces are grayish spots of fibrinous exudate in which are small black charred particles. On the edges of the fibrinous membrane focal hemorrhage can easily be made out. The hemorrhage and discoloration are present in the larynx below the vocal cords as well as above them. As one passes down the trachea intense hemorrhagic congestion continues but the fibrinous membranes disappear except for scattered linear erosions 2 to 4 mm. in length and 1 to 1.5 mm. in width. The tracheotomy wound is clean and shows no evidence of infection. All the tissues of the lower neck are markedly edematous and this edema spreads downward over the anterior chest wall beneath the pectoral muscle and also down into the anterior mediastinum in the region of the thymus.

**Lungs** Combined weight 1630 Gm. When examined *in situ* it is evident that they collapse only slightly on removal of the sternum. The anterior margins of both upper lobes which present through the sternectomy incision are light in color and the borders are rounded. Atelectasis is clearly visible to the naked eye indicating physiologic emphysema. The major portions of both upper lobes and of the middle lobe are dark pink with a slight violaceous tinge. The tissue feels heavy and is of increased consistency but, nevertheless still crepitant. No areas of true consolidation can be felt. Two-thirds of the left lower lobe is similar in consistency whereas the posterior inferior third is completely atelectatic and dark reddish-blue and moderately firm. The right lower lobe is likewise heavy with diffusely decreased crepitation except for a transverse band of complete atelectasis 5 cm. in width, the lower border of which lies 4 cm. above the diaphragmatic margin. Section of the unfixed right lung demonstrates extensive membranous bronchitis extending even to the bronchioles which are dilated and can be traced to within 0.5 cm. of the pleural surface. The membrane is less adherent than in Case 25 is more mucoid and more often stained by hemorrhage. The cut surface of the lung is deeply congested, slightly firmer than normal and diffusely subcrepitant. Moderate amounts of fluid can be expressed with pressure. In the middle third of the lower lobe it is completely atelectatic, very dark red and moderately firm. Several small frankly hemorrhagic foci, usually lobular in dimensions are found in contact with pleural surfaces. These suggest infarction but are less firm than would be expected.

Section of the left lung after fixation shows a uniform reddish-chocolate color, alveoli which are just visible to the naked eye and no variations in consistency. The lower lobe in contrast shows marked variations. An irregularly pyramidal area, with a base 4 cm in diameter along the diaphragmatic surface, extends upward in a zig-zag pattern along the posterior surface of the lobe for a distance of 11 cm. It is dark red in color and depressed 2 to 3 mm below the adjoining parenchyma. In the fixed specimen it is extremely firm. The cut surface is, likewise, dark red in color, frankly hemorrhagic and completely homogeneous. No air bubbles are present and no trace of alveolar markings can be discerned. At its upper margins the hemorrhagic process sends finger-like processes toward the hilus which are localized in the adventitia of large vessels and bronchi. The configuration of the lesion is very characteristic of atelectasis but its consistency and appearance on section is that of infarction. The remainder of the lower lobe shows compensatory physiologic emphysema, the alveoli averaging nearly twice the size of those in the upper lobe.

*Mediastinum* The tissues of the anterior mediastinum are distinctly congested and edematous. As mentioned above this appears to be a dependent drainage of fluid from the more massive edema in the lower neck.

*Liver* Weight, 2265 Gm. It seems large even in proportion to the patient's size. It is, in general, deep reddish-brown with some focal yellowish mottling. On section, the markings are not prominent, and the tissue does not pout over the cut edge.

*Spleen* Weight, 260 Gm. It is very firm, intensely congested. Considerable pulp scrapes readily from the cut surface. The markings are not evident.

*Adrenals* Normal.

*Kidneys* Weight, 370 Gm. They are intensely congested. The capsules strip readily leaving a smooth surface. The cortex averages 6 mm in thickness. The pelves show scattered minute petechial hemorrhages.

*Microscopic Examination*—*Larynx and Trachea* The simplest change is a loss of the lining epithelial cells which have been replaced by a fibrinous membrane of varying thickness in which black granules, presumably carbonaceous, are embedded. This fibrinous layer appears loosely adherent and has frequently been detached in the process of sectioning. Beneath these areas the basement membrane is intact, the underlying areolar tissue is edematous but contains no fibrin deposit and comparatively little leukocytic infiltration. Where epithelium persists it is represented by a narrow layer, one or two cells thick, of spindle-shaped basophilic cells, the long axes of which parallel the basement membrane. Though no mitoses are found one may legitimately surmise early regeneration.

In the more severely involved areas a different picture is observed (Fig 29). The mucosal surface is lined by a necrotic layer in which two zones can usually be recognized. Toward the lumen is a hyaline, brightly acidophilic, sometimes "fibrinoid" zone 50 to 100 microns in thickness, which is usually devoid of any recognizable structural pattern and is cell free except for small numbers of infiltrating leukocytes. In a few areas, however, shadow forms of the original cylindrical ciliated cells can be recognized within it, suggesting that in part at least this hyaline zone has formed by necrosis and fusion of the epithelial layer. Beneath this hyaline zone is another zone, 100 to 200 microns in thickness, of true fibrin deposit and dense leukocytic infiltration. This zone appears to lie just beneath the basement membrane, wherever remnants of it can be identified.

All the blood vessels of the mucosa appear maximally dilated and engorged. A number of the superficial vessels close to the fibrinous layer just described contain thrombi which partially or totally occlude the lumina. In the smaller vessels closest to the surface these thrombi are often dense and hyaline, in the larger ones they are of ordinary platelet and fibrin structure. The loose connective tissue of the deeper portions

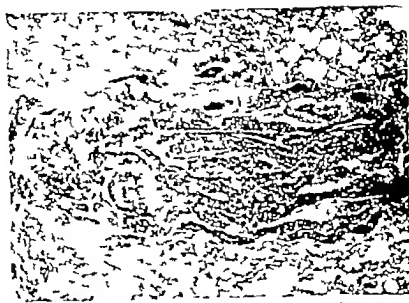


FIG. 28. — Small bronchus from Case 25 completely occluded by a plug made up of desquamated necrotic epithelium, leukocytes, fibrin and mucin.



FIG. 29. — Segment of trachea just beneath the larynx from Case 7. The epithelial layer and basement membrane have been replaced by a hyaline fibrinous membrane. The underlying mucosa is thickened by edema.



FIG. 30. — A tertiary bronchus from Case 7. The epithelial layer is still in situ but completely necrotic and partially hyalinized. On the left margin small lakes of serous exudate present between the necrotic layer and the basement membrane. It is believed this represents the mechanism of spontaneous detachment of the necrotic tissues.

of the bronchial wall is markedly edematous and contains small numbers of scattered polymorphonuclears

*Bronchial Tree* The epithelial layer where visible is completely necrotic. In other areas it has desquamated leaving a denuded but intact basement membrane. Over considerable distances the shadow forms of the necrotic epithelial cells are visible as a distinct layer, sometimes still adherent to the basement membrane, more often partially separated from it by small lakes of serous exudate (Figs 30 and 31). The lumina of most of the larger and all the smaller bronchi are plugged with casts composed of necrotic desquamated epithelium, polymorphonuclear leukocytes, monocytes often containing black granules, fibrin and traces of mucus in varying proportions. One bronchus contains a mass of cornified epithelial cells.

The deeper layers of the bronchial mucosa show maximal vascular engorgement and in some instances extensive interstitial hemorrhage. This frequently extends into the adventitia external to the cartilaginous rings. Leukocytes are usually concentrated just beneath the basement membrane and are scanty in the deeper layers.

*Pulmonary Parenchyma* The picture varies widely in different portions of the lung. Throughout most of the lung the picture is one of physiologic emphysema alternating with edema and partial collapse. In the latter areas the alveoli average one-third to one-half the size of expanded alveoli, and are uniformly fluid-filled. No complete alveolar collapse is found. Even in the partially atelectatic areas, foci of trapped air are found in overdilated atria and respiratory bronchioles. The edema fluid for the most part has precipitated as an homogeneous, colloid-like mass in the alveoli. There are no fibrinoid membranes and little diapedesis of red cells.

Sections from the large hemorrhagic area (Fig 32) described at the base of the left lung are somewhat unusual in appearance. The alveoli are uniformly small, one-third to one-half the size of those in neighboring areas. Their lumina are uniformly devoid of air as are also those of the atria and respiratory bronchioles. The majority are packed with red cells but others contain colloid-like edema fluid, and still others interlacing strands of fibrin, and all combinations of these three elements occur. Almost no leukocytes are present. The alveolar walls are perfectly preserved, their nuclei stain well and their capillaries can usually be made out and appear congested. The adventitia of all blood vessels in the zone is markedly hemorrhagic and packed red cells are found in many lymphatics.

Scattered throughout all lobes of both lungs, nonadherent hyaline emboli, similar to those noted in the superficial vessels of the larynx and trachea, are found in many of the pulmonary arterial branches. Other vessels, in the neighborhood of the hemorrhagic areas, show fresh, occlusive thrombi of the usual platelet and fibrin type.

*Liver* Moderate fine, fat vacuolization is present in the hepatic cells of the central third of the lobule. In this same region slight edematous separation of the sinusoidal endothelium from the liver cords is evident.

*Kidney* Negative

*Adrenal* The cell cords in the outer half of the fascicularis and inner glomerulosa show separation of the cells by pools of serous exudate in which a few strands of fibrin are frequently found. In an occasional focus there is complete interruption of the continuity of the cord where cells have completely disappeared. An occasional brightly eosinophilic cell with pyknotic nucleus and a few foci of polymorphonuclear infiltration are found (Fig 33).

*Stomach* A section from the fundus shows marked vascular engorgement and scattered petechial hemorrhages in the mucosa.

*Brain* There is disorganization of the Nissl substance of the cortical nerve cells. Scattered astrocytes in the cortex have swollen cytoplasm and two or three nuclei.

FIG. 31.—Higher magnification of portion of the wall of the same bronchus showing the partially fused necrotic epithelial layer and the inflammatory reaction in the deeper layers of the mucous membrane.



FIG. 32.—Edge of the large depressed hamorrhagic area in Case 7, simulating infarction but showing no necrosis of alveolar walls.



FIG. 33.—Fascicular layer of the adrenal cortex of Case 7. The cell cords are disrupted by accumulation of fluid and a small collection of polymorphonuclear leukocytes enmeshed in threads of fibrin.



Marked satellitosis, such as is present in Case 27, is not observed, but sections from a corresponding region of the brain are not available

Case 7, in summary, showed essentially the same type of membranous necrotizing inflammation of the larynx, trachea and bronchial tree as Case 25. In comparison, the process was somewhat more severe in the larynx (tracheotomy had been performed) and a little less intense in the bronchial tree. In contrast, the pulmonary changes were radically different. Whereas pre-existing anatomic emphysema and acute pulmonary edema dominated the picture in Case 25, in this one, atelectasis and compensatory physiologic emphysema were the rule. A complicating factor was provided by the extensive hemorrhagic lesions in the lower lobes. These are extremely difficult to interpret. Their gross configuration and their distribution conformed with the usual pattern of atelectasis, but the character of the cut surface seemed typical of infarction. Microscopic examination showed extensive hemorrhage into the alveoli compatible with infarction and multiple arterial emboli. However, the alveolar walls showed no necrosis and there was no trapped air within the lesion. No leukocytic infiltration was present, even at the margins.

The problem is to explain the massive alveolar hemorrhage. The picture was clearly not pneumonic. Blood aspiration from massive bronchial hemorrhage ordinarily presents a dispersed, fan-like pattern rather than the massive involvement present here. Furthermore, no clots were present in the bronchial tree and there was no history of massive hemoptysis. Simple atelectasis,<sup>1</sup> when based on bronchial occlusion, not on external pressure, is associated with serous exudate into the alveoli and moderate numbers of red blood cells may be present, but they never dominate the picture.

The final alternative is infarction. In very recent infarction sufficient time may not have elapsed to produce visible changes in the alveolar walls but in such early infarcts trapped air is almost invariably found. A condition known as partial infarction<sup>2</sup> must also be considered. This is a parenchymal injury based on arterial occlusion which does not go on to tissue necrosis because a collateral circulation develops within a comparatively short period. In this condition extensive hemorrhage into alveoli may occur, but again total displacement of air would be most unlikely. The final possibility to be considered is that infarction developed in a previously collapsed atelectatic area. This hypothesis would fit the gross appearances, but since the microscopic examination failed to show evidence of necrosis of alveolar walls it would be necessary to assume that it was very recent or that the infarction was but partial. The evidence does not warrant a definite diagnosis.

Another feature of note was the microscopic demonstration of adrenal cortical necrosis. This is of interest because of the physiologic evidence of change in adrenal cortical function obtained by Dr. Cope, and his associates, from study of the 17-ketosteroids excretion (metabolic observation). The brain showed histologic changes characteristic of anoxemia.

Case 27—The body is that of a well-developed and well-nourished young woman age 18. There are extensive superficial burns as follows: Third degree burns, the

right side of the face, the forehead and the mouth. There is also a spot of third degree burn, 2.5 cm. in diameter over the left malar bone. The margins of both ears are deeply burned and there is a second degree burn on the upper lip and most of the chin. Third degree burns cover the dorsum of both hands extending 4 cm. above the wrists and also the lateral aspect of both upper arms from elbows up to the acromion processes. The entire back from the buttocks up to the shoulders is completely covered by second and third degree wet, weeping lesions. Small scattered burns, each about 1 cm. in diameter are found scattered along the anterior aspect of both lower legs overlying the tibial crests.

*Trachea and Bronchi* Intense congestion is present without visible necrosis. There are scattered petechial hemorrhages in the upper trachea.

*Bronchial Lymph Nodes* Slightly enlarged and wet.

*Lungs* (color section Fig. 8) Weight 1180 Gm. The lungs fail to collapse when the sternum is removed. Examined *in situ* the lower two thirds of the left lower lobe is markedly atelectatic. The remaining portion of the lobe shows compensatory emphysema interrupted by a few scattered lobular areas of atelectasis. One transverse band of atelectasis about 1 cm. in thickness traverses the upper portion of the lobe at a level 5 cm. below the apex. Another band runs vertically at right angles to this up to the extreme tip of the lobe. On the lateral surface of this lobe adjoining the diaphragmatic margin is a purple area of sharply outlined consolidation 3 cm. in diameter over which petechial hemorrhages are found in the pleura. One centimeter posterior to this is a smaller similar nodule, 1 cm. in diameter. The left upper lobe is again predominantly emphysematous with readily visible alveoli but shows several scattered small patches of atelectasis ranging from 1 to 2.5 cm. in diameter. The right lower lobe shows massive atelectasis of the posterior two-thirds, whereas the anterior third is distended except for occasional purple lobular areas of collapse. A roughly spherical nodule of consolidation, 3.5 cm. in diameter is present in the atelectatic portion on the lateral surface of the lung 2 cm. above the diaphragmatic border. The right middle lobe is predominantly emphysematous with a focus of increased consistency near the lower anterior margin 3 cm. in diameter. This is light purple in color there is no overlying pleural reaction. The right upper lobe is markedly emphysematous with a few small consolidated nodules about 1 cm. in diameter along the anterior margin.

A sagittal section of the right lung (color section Fig. 9) reveals marked congestion of the mucosa of the primary bronchus. Beginning in the lobar bronchus and becoming more intense in their secondary and tertiary subdivisions there is mucosal hemorrhage necrosis and pseudodiphtheritic membrane formation which extends even into the small bronchial radicles (color section Fig. 7). Large areas of red, depressed atelectatic tissue alternate with pale aerated slightly overdistended parenchyma. Three sharply outlined foci, ranging from 1 to 3 cm. in diameter extend inward from the pleura, which are deep red in color very firm to palpation and have the gross appearance of infarcts. Thrombosed vessels are found in association with two of these.

Section of the fixed left lung shows essentially a similar picture. One area of apparently typical infarction is found on the diaphragmatic border. Approximately one third of the upper lobe and two-thirds of the lower lobe are partially to completely atelectatic. In some areas the atelectatic tissue is firm enough to raise the suspicion of early consolidation.

*Microscopic Examination—Trachea and Bronchi* An extensive superficial necrotizing process is present which never extends beneath the basement membrane. No normal epithelium is found. In a few areas necrotic shadow forms of epithelial cells lie in their normal positions still attached to the basement membrane. Frequently small pools of serum are found between the basement membrane and the necrotic layer and in these areas all stages of desquamation are apparent. In many areas no traces of epithelium are left and the basement membrane is either bare or covered with a fibrinous



membrane containing numerous leukocytes. Interspersed with such areas are others where variable proportions of epithelium persist. Occasionally it is of nearly normal thickness but without recognizable differentiation into ciliated cells or goblet cells. Marked leukocytic infiltration is invariably present between the persisting cells. More commonly, the epithelial layer is thin and consists of one, two, or occasionally three layers of somewhat elongated oval or spindle-shaped cells whose long axes parallel the basement membrane (Fig 34).

The deeper layers of the bronchial walls are invariably congested and occasionally hemorrhagic. The degree of leukocytic infiltration is not, however, great except for a narrow zone immediately beneath the basement membrane. Blood vessels within the bronchial walls are not thrombosed. The lumina of all medium-sized and small bronchi are more or less completely plugged with exudate. This consists in part of desquamated epithelium, in larger part of fibrinopurulent exudate intermixed with some masses of mucin. In one bronchus masses of cornified epithelial cells are found, presumably aspirated from higher in the respiratory tree. In another bronchus two large clumps of micrococci are seen.

*Pulmonary Parenchyma* The picture is variegated, being compounded of intermixed atelectasis and physiologic emphysema, edema, pneumonia and focal infarction.

The edema is for the most part associated with partial atelectasis. The alveoli are smaller than normal but never completely collapsed and their lumina contain extensive granular deposits. In one respect, however, the picture varies from the preceding three cases. In many areas a brilliantly acidophilic fibrinoid membrane has formed peripheral to the granular material and in apposition with the wall of the air passage. These membranes are usually found in respiratory bronchioles and atria and comparatively rarely in alveoli (Fig 35). In a few areas clusters of red blood cells are mixed with the granular precipitate.

The areas of infarction are lobular in size and the alveolar lumina are solidly packed with red cells. The alveolar walls are largely viable but in the center of the lesions they show early signs of necrosis. No trapped air is found in these foci, and bronchioles extending into them show no necrosis of their walls. One focus of partial infarction is found in which the alveolar lumina are packed with red cells and the capillaries contain hyaline thrombi but the alveolar walls are not necrotic. Throughout the lung, sometimes in comparatively normal areas, small, dense, usually nonocclusive thrombi are present in occasional vessels (Fig 36). Two fresh fibrin and platelet thrombi are found in vessels 2-3 mm in diameter. These are adherent to the walls and one shows traces of organization at the periphery.

The pneumonic foci (Fig 37) tend to be peribronchial in one location. They show serous precipitates, fibrin, red cells and numerous polymorphonuclear leukocytes. In some of the pneumonic foci, fibrinoid membranes are present and, again, are usually found in respiratory bronchioles and atria rather than in alveoli. Organisms are not numerous and are entirely intracellular. They appear to be cocci in pairs and short chains and are never lanceolate.

*Adrenals* Distinct abnormalities are present. The cell cords, particularly in the outer half of the fascicularis, are frequently split and sometimes partially transected by spaces in which granular detritus has precipitated, indicating an accumulation of serous fluid. In these spaces mononuclear phagocytes are present in small numbers. In a few small and scattered foci acidophilic necrosis of adrenal cells is present and in these areas polymorphonuclears have collected (Fig 38). The chromaffin cells of the medulla appear shrunken and the space between them and the sinusoidal endothelium is widened, suggesting edema.

*Brain* The cerebral cortex is definitely abnormal. Many of the larger nerve cells including some of the Purkinje cells are surrounded by an increased number of satellite cells. Several mitotic glia cells are observed. Oligodendroglia cells in the

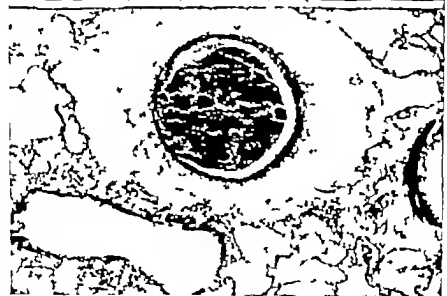
FIG. 34. — A bronchus from Case 27. The lumen is plugged by desquamated cells, fibrin and leukocytes. For the most part the basement membrane is dotted but on the left some reappearing atypical epithelial cell can be seen.



FIG. 35. — Case 27. Partially collapsed alveolus containing serous precipitate and a few leukocytes. The respiratory bronchioles are lined with an adherent fibrinoid membrane.



FIG. 36. — Case 7. An arteriole from a normal portion of the lung showing a nonadherent rather hyaline embolus occlusion. The perivascular lymphatic is widely dilated. Similar emboli were numerous in Case 7.



the victims had inhaled an irritant agent, either physical or chemical, they had lived long enough to develop a serous but not a leukocytic reaction and that, therefore, their survival period was to be measured in minutes rather than in seconds or in hours. The mechanism of death was anoxic dependent in part upon inhalation of carbon monoxide and in all probability other gases, and in part upon edema of the lungs.

The cases which died after varying periods upon the wards showed a similar distribution and extent of cutaneous burns. The major visible lesions were again found in the respiratory tract. In Cases 7 and 25 se-



FIG. 40—A higher magnification of the bronchial cast seen in Figure 39. Note the necrotic respiratory epithelium in the separated membrane.

necrotizing laryngitis, with the formation of a pseudodiphtheritic membrane had led to almost complete laryngeal stenosis. It is interesting that the process was most severe beneath, rather than above the vocal cords.

All three cases showed diffuse hemorrhagic and focal membranous reaction in the lower trachea and primary bronchi. All three showed diffuse membranous bronchitis most severe in the secondary and tertiary subdivisions of the lobar bronchi but extending in many areas to the small bronchioles. The membranes were found to be sometimes firmly adherent to the bronchial walls, sometimes readily separable. In many areas, notably in Case 7, the membranes had spontaneously separated and had coiled themselves within the bronchial lumina to form occlusive plugs. One of these which was spontaneously raised by a patient who survived, is illustrated in Figures 39 and 40.

Microscopic examination showed diffuse necrosis of the lining epithelium. In many areas the shadow forms of the necrotic cells rested *in situ* on the intact basement membrane. The accumulation of small lakes of serum between this layer and the basement membrane gave a clue to the mechanism of spontaneous detachment. In other areas all traces of the epithelium had disappeared and a dense rather hyaline fibrinous membrane rested upon an intact basement membrane. In the larynx and trachea but rarely in the bronchi evidence of deeper necrosis was found and the basement membrane was itself partially or completely destroyed. Even in these areas the zone of necrosis was not deep never wider than 100 microns. In Case 7 many of the vessels in and just beneath this zone were thrombosed.

In contrast to the fairly uniform changes in the tracheobronchial tree the pulmonary lesions varied considerably from case to case. Case 7 a male age 29 showed severe anatomic emphysema, evidently of many years standing. Superimposed upon this were an acute pulmonary edema and the barest traces of early bronchopneumonia. Case 25 showed extensive atelectasis and compensating physiologic emphysema. A complicating factor which is difficult to interpret was the presence of massive hemorrhage into the alveoli of large atelectatic areas grossly suggesting infarction but microscopically showing no necrosis of alveolar walls. The presence of multiple miliary emboli throughout the pulmonary arterial tree provided the necessary precipitating factor and it is suggested that preexisting atelectasis may have been the substrate. In the aerated portions of the lung in contrast no reaction to the presence of the emboli could be detected. Case 27 was in many respects similar. Again atelectasis and compensatory physiologic emphysema dominated the picture. In this case too multiple small emboli were found throughout the lung, in normal as well as in abnormal areas. Small foci of hemorrhage usually only of lobular dimensions were present some of which were typical both grossly and microscopically of infarction others showed viable alveolar walls like the lesions in Case 7. A further complicating factor was the presence of widespread foci of incipient bronchopneumonia within the areas of atelectasis. The very slight solidification of the lungs on gross examination the predominant serous exudation and the comparatively light leukocytic infiltration all suggest that this pneumonic reaction was of very brief duration.

Other organs with few exceptions showed no abnormalities which could be attributed to the acute incident. The liver in Case 25 was markedly infiltrated with fat and that in Case 7 moderately so. In both instances the habitus of the patient suggested that its presence was physiologic. Careful examination of the kidneys showed no evidence of hemoglobinuric nephropathy though this was found in several cases examined elsewhere in the city. The gastro-intestinal tract regularly showed congestion and petechial hemorrhages in the fundic portion of the stomach and in the duodenum.

One parenchymal lesion could it was believed clearly be attributed to the acute injury. This was focal necrosis of the adrenal cortex very

clearly demonstrable in Cases 7 and 27. In two cases the brain showed changes attributable to prolonged anoxemia.

## DISCUSSION

Many of the facts of the Cocoanut Grove disaster are still shrouded in mystery, and it is not improbable that some of them will always remain so. It is clear that the victims were exposed briefly to flame of great intensity arising from inflammable material and, for a longer period, to toxic products of combustion which, in the enclosed space, may have reached high concentrations. Of the nature of the latter, nothing as yet is definitely known, beyond the demonstrated presence of carbon monoxide and at least traces of oxides of nitrogen. It may be assumed that both factors may have played a rôle in the pathologic lesions which were observed.

Testimony indicates that the flames were, to a considerable extent, limited to the upper portions of the various rooms. This checks with the pathologic observation that the heads of the victims were disproportionately burned. It also makes it understandable that inhalation of flame was frequent. How far flames may have penetrated into the respiratory passages is more difficult to determine with precision. Characteristic eschars were found within the nares, the black membranous exudation of the larynx, and the lesions in the trachea were probably of similar origin. The exaggeration of the necrotizing process below rather than above the vocal cords was presumably due to eddy formation below a point of narrowing. Lesions of this type are not unusual in the experience of a Medical Examiner.

More difficult to account for was the massive pulmonary edema which must have developed in an extremely brief period of time. Even with the most toxic war gases some latent period is usual, frequently a period of several hours. Anoxemia alone will rapidly produce pulmonary edema as Drinker, and his collaborators<sup>3</sup> have shown. Perhaps a combination of anoxemia and some irritant vapor may have worked synergistically in these cases.

The most characteristic feature of the material was the diffuse membranous bronchitis. This is a characteristic effect of several of the war gases, notably phosgene, mustard gas and chlorpicrin.<sup>4</sup> It is also characteristic of nitrogen dioxide inhalation.<sup>5</sup> Herein lies the strongest pathologic evidence for inhalation of toxic fumes.

## REFERENCES

- <sup>1</sup> Loeschke, H. Atelektase. Henke-Lubarsch Handbuch der speziellen Pathologischen und Histologie, Vol. III, Part I, page 603.
- <sup>2</sup> Hampton, A. O., and Castleman, B. Correlation of Postmortem Chest Teleroentgenograms with Autopsy Findings. *Am J Roentgenol and Radium Therapy*, 43, 305-325, 1940.
- <sup>3</sup> Warren, M. F., Peterson, D. K., and Drinker, C. K. Effects of Heightened Negative Pressure in the Chest Together with Anoxemia in Increasing the Flow of Lymph from the Lungs. *Am J Physiol*, 137, 641-672, 1942.
- <sup>4</sup> Wintermiz, M. C. Pathology of War Gas Poisoning. Yale University Press, 1920.
- <sup>5</sup> von Ottingen, W. F. The Toxicity and Potential Dangers of Nitrous Fumes. *Public Health Bulletin No. 272*, 1941.

# THE TREATMENT OF THE SURFACE BURNS\*

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UNTIL THE INTRODUCTION of physiologic methods of investigation into the clinic, the treatment of burns was merely the treatment of the surface wound. This was inevitable since the wound was obvious to physician and patient. In the last 50 years with the recognition of hemoconcentration the danger of the generalized shock of burns has been appreciated and an argument has developed within the profession as to which takes precedence in the treatment the care of the surface wound or the care of shock. Even recently men primarily interested in the shock picture say that nothing should be done to the surface until impending shock has been prevented or existing shock adequately treated.

Such emphasis on shock to the neglect of the surface treatment is wise if the surface treatment is one which leads to further shock. Débridement and cleansing under anesthesia is such a treatment.

Delay in the care of the surface wound, however, inevitably leads to bacterial contamination. With increase in contamination there is increased infection, and increased infection leads to shock as well as delayed wound healing.

Overemphasis on the surface treatment to the neglect of that of shock and anoxia and a tendency to attribute special virtues to a surface treatment are also unenlightened. For example when 'toxins' were considered an etiologic agent in shock tannic acid was advised on its presumed ability to fix *in situ* tissue toxins produced by the burn<sup>1</sup> and reduction in the mortality of patients with burns has been ascribed to a surface treatment whereas in reality it was due to better care of early shock.

The surface treatment cannot be divorced from the treatment of shock. It is the purpose of this article to outline a surface treatment which does not interfere with the life saving treatment of shock yet tends to prevent bacterial contamination. This treatment is peculiarly suited to a catastrophe where the large number of burn casualties is out of proportion to the number of trained personnel. The problem of the therapy of shock is considered in a subsequent article.

## THE SURFACE TREATMENT USED ON COCOANUT GROVE PATIENTS

Thirty nine of the 114 patients brought to the Emergency Ward of the Massachusetts General Hospital from the Coconut Grove night club fire survived the initial few minutes and received treatment of their burns. Common to disasters there was a pattern to the injuries. The burn pattern consisted of the hands, face, nostrils, mouth, the lower half of both cheeks and the scalp and in the women areas unprotected by adequate clothing, namely the neck, arms, shoulders, back and legs. In addition to the pattern distribution the back and legs of some of the men were burned. Burns

\* The work described in this paper was done under a contract, recommended by the Committee on Medical Research, between the Office of Scientific Research and Development and Harvard University.

of all degrees were encountered, many having third degree or full-thickness burns

The burns were sooty and shaggy, with unruptured blebs. Many were cherry-red color from carbon monoxide poisoning. Some of them looked clean, others grossly dirty. Many of the patients had been crawling on the floor at the fire, others had been dragged out from piles of dead. Many of the dead, and some of the unconscious who lived had been incontinent of both urine and feces.

But one type of surface treatment was applied to the burns of the skin of all of the 39 patients. A bland ointment with protective dressing was applied without any preliminary débridement or cleansing, and antibacterial chemotherapy was given internally.

As the patients entered the Emergency Ward, either walking or on a stretcher, sterile towels were placed over the burned surfaces. No covering was put over the faces. Insofar as possible these towels were held in place as the patients were undressed and transferred from stretcher to bed. For those patients having burns of the back, buttocks, and upper legs, sterile sheets were placed on the bed.

A needle was inserted for the intravenous administration of plasma before the dressings were applied to the burned surfaces. In many this was done while the patient was still on the stretcher.

When a patient was settled in bed, the sterile towels were folded back and the burn surfaces covered with sterile boric ointment strips. These strips were applied by interns or medical students who were not only capped and masked but were scrubbed and wearing rubber gloves. *The burn surfaces were neither cleansed nor débrided* \*

The boric ointment gauze of fine mesh was covered with sterile gauze to protect the wounds. Burns of the face, scalp, and of the extremities were bandaged with pressure. This was accomplished by adding roller elastic bandages on top of the gauze dressing. Over the burns of the trunk, large stockinet rolls were applied. The dressings of burns of the neck were held in place by ordinary gauze bandages. The eyes, after application of 5 per cent sulfathiazole ointment, were closed and were included in the pressure dressings of the head. Only the nostrils and lips were left uncovered.

As a final part of the initial surface treatment, two grams of sodium sulfadiazine were injected intravenously through the cannula or needle already in place for plasma transfusion.

This surface dressing, and the chemotherapy, completed the care of the wound. It was first aid and definitive treatments combined. The dressings were not changed until the fifth to tenth day, when boric ointment gauze was reapplied. To those burns which proved to be of second degree no other type of dressing was applied. To those areas later proving to have deep de-

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\* The face and left hand of one patient (Case 17) was partially débrided and cleansed with soap and water before applying a dressing of 5 per cent sulfathiazole ointment. This was done before the general order was appreciated.

struction of the skin wet dressings of boric acid or physiologic saline solution were used after the first two weeks in order to expedite the removal of the burned tissue, and prepare the surfaces for grafting

*Rationale of the Surface Treatment Employed*—The use of this orthodox surface treatment was premeditated and prompted by the concept that of treatments giving approximately equal results the simplest would be the one best adapted for a disaster with numerous burn casualties

*No Débridement and No Cleansing*—We did not debride the burn wounds because we were convinced that the intact epidermis over the blebs protects against the entrance of bacteria and because bleb fluid does not become contaminated by virulent organisms harbored in gland crypts

We did not cleanse the burn surfaces because we believe that cleansing is ineffectual in reducing significantly the number of contaminating organisms present unless vigorous scrubbing is resorted to. Such scrubbing it is believed injures viable epithelium. An anesthetic also would be necessary and is undesirable from the point of view of augmenting shock.

The débridement of burn wounds as commonly carried out consists not only of picking off particles of clothing and other foreign material but also in rupturing any vesicles and removing the overlying epidermis and all of the loose epidermis of the vesicles already ruptured. This is ordinarily followed by cleansing. Admittedly débridement is necessary for good tanning and therefore débridement and cleansing are part of the tannic acid ritual they have been carried over into other methods of treatment without criticism. These maneuvers are time-consuming as well as painful. They tie-up available personnel and require analgesic drugs if not general anesthetics. The increased manipulation of the patient prolonged exposure of the wounds and anesthetics attendant with débridement are conducive to serious shock. If these maneuvers can be eliminated the trained personnel will be freed and the prevention of further shock accomplished.

When nondébridement of burn wounds was suggested soon after Pearl Harbor serious objections were raised. It was maintained that all blebs must be opened because bleb fluid would be an excellent anaerobic culture medium and because the bacteria harbored in the gland crypts at the base of the vesicle would swim up and infect the fluid.

On physiologic grounds the first objection seemed unreasonable. Superficial human burn wounds weep for many hours and in dogs Field, Drinker and White<sup>2</sup> in 1931 showed that there was an increased flow of lymph induced by an experimental burn which continued for hours without clotting. These observations suggested a rapid turnover in the protein rich fluid in the burn areas. The fluid comes from the plasma pouring through the open capillary membrane into the extracellular spaces and passes either out on to the surface or back through the lymphatics. Such a rapid turnover of plasma fluid should mean a relatively high oxygen content of the edema fluid in the burn area.

Against the second objection are the bacteriologic observations of Colebrook<sup>3</sup> and others<sup>4,5</sup> pointing to the ability of skin to rid itself of patho-



genic bacteria. There are normal habitants of skin which are not sufficiently virulent to cause infection, and little trouble is to be expected from these. Virulent bacteria are destroyed, perhaps by the skin lipoids<sup>5</sup>. The heat producing the burn also decreases the number of bacteria present on the skin at the time.

Finally, it remained to find out in patients with burns what happened to the fluid of, and healing beneath, the unruptured vesicles. Twenty-six patients had been studied prior to the Cocoanut Grove fire. The fluid from more than one bleb was observed in many of the patients. In those arriving at the hospital with unruptured blebs, the blebs were protected by gauze to prevent rupture. Many of the patients' burns were hours to days old when first seen, and the blebs had not been protected from contamination by any dressing. From 24 hours to 14 days after the burn the fluid was removed from the vesicles under sterile precautions and cultured in Dr. Champ Lyons' laboratory. In a few instances, the nonpathogenic saprophytic organisms of normal skin were recovered. In only one instance was a pathogenic organism, a *beta* hemolytic streptococcus, obtained in fluid of an unruptured bleb, and in this case alone was the fluid purulent. It was the clinical impression that the healing beneath the unruptured blebs occurred as rapidly as under any of the agents commonly recommended for the burn surface.

Another objection which has been raised to *nondébridement* is that the dead epidermis of vesicles which have already ruptured provides a culture medium for organisms. Such broken epidermis usually retracts and is often found rolled up in a corner of the old vesicle. Admittedly, it looks messy and unsurgical, but how dangerous a culture medium is it and what do we accomplish by taking it away? If it is excised and a dressing applied, it will be replaced by a layer of fibrin between cells and dressing. As far as is known, this inert fibrin is as good a culture medium as the dead epidermis. Since unruptured vesicles are not infected, the under surface of the broken epidermis is presumably uninfected. Even though it now covers a lesser surface of the burn, it is still a protection and as physiologic as any that is now known.

A major point in favor of *nondébridement* of the burn wound is, as has been shown in this laboratory, the availability to the wound of chemotherapeutic agents administered internally. Absorption of sulfonamides applied locally to the débrided burn surface has been observed at this hospital by Dr. Lyons, and also reported in the literature. Such absorption may be rapid and irregular and if the burn surface is large, toxic levels of the drugs in the body fluids may be reached. (Absorption from a *nondébrided* surface has not been measured.)

The levels of the sulfonamide drugs in the body fluids are more easily controlled by internal administration. Therefore, if it could be shown that these drugs permeate through the burn tissue, this route would be preferable. Since sulfonamides are freely diffusible and there is an increase in capillary permeability in the region of the burn with delayed clotting of the edema

and bleb fluid it seemed likely that free diffusion in the early hours at least would be found.

As anticipated the level of the sulfonamides in the bleb fluid of unruptured vesicles followed closely that of the blood plasma. For example a girl with a burn of the leg and a large unruptured bleb was started on sulfadiazine by mouth two hours after the burn. Twelve hours later a titer of 4.3 mg of the drug was found both in the bleb fluid and blood plasma. Such observations were repeated on a number of the Coconut Grove fire patients.

It is not known for how long sulfonamides administered internally permeate freely through the burn wound. Fibrin is eventually deposited in the intercellular spaces of the wound and it is probable when this stage of the inflammatory process is complete that substances normally diffusible are no longer able to permeate through the wound in bacteriostatic concentrations. This is suggested by the findings in a patient treated since the Coconut Grove disaster. A hot water burn of the lower leg resulted in a large unruptured bleb. Sulfadiazine was withheld until 60 hours after the burn. At 73 hours the levels of sulfadiazine were 8 mg in blood and 3.3 mg in the bleb fluid. The time limit if such exists remains to be determined.

It seemed reasonable on the basis of these findings on the fluid of unruptured blebs (absence of virulent bacteria, good healing in its presence and availability to it of internally administered sulfonamides) to treat burn patients with the greatly simplified surface treatment of no débridement, no cleansing and a simple bland ointment with protective dressing. Several patients had been treated in this manner prior to the Coconut Grove disaster and the observations on them afforded an adequate basis for planning to use such a simplified treatment in a disaster with numerous burn casualties.

*Boric Ointment.* A bland protective ointment dressing is indicated in the treatment of skin burns since the chemical agents currently recommended are believed to be injurious to otherwise viable epithelium and delay wound healing. In a previous communication from this hospital<sup>6</sup> it has been shown that tannic acid, the dye solutions and certain other preparations delay the healing of an epithelial wound. Use was made of the donor site, from which a skin graft of uniform thickness had been removed by the dermatome for the assay of these various agents. This wound heals by epithelial proliferation with a minimum of fibrous tissue contracture. It is also a sterile wound and the retarding influences of various infectious organisms is eliminated as a complicating factor. Although it is not a burn wound and does not have the superficial layer of dead tissue created by the burn, from the point of view of healing it has much in common with the burn wound. Substances retarding healing in this donor site wound would presumably have the same action on the viable epithelium of a burn wound.

It is difficult if not impossible to assay accurately the effect of a chemical agent on epithelial regeneration using a clinical burn wound. There are factors other than the substance applied locally which influence

the rate of healing There is no proven clinical method of judging accurately the extent to which the cells are damaged by the burn, and thus no two burned areas can be considered of identical depth or degree Obviously, the deeper the burn, the slower the healing will be Also complications of the burn, infection and malnutrition, delay the eventual healing The effect on healing ascribed to an agent may be due in reality to the depth of the burn or to other factors \*

Boric ointment gauze was used as the control agent in these experiments upon epithelial regeneration It is chiefly because this ointment is commonly used in hospitals for many purposes that it has been chosen for treating burns Dr Lyons feels that the boric acid in the ointment may inhibit the growth of the pyocyanus organism (It is known to rid granulating wounds of pyocyanus infection) The relative absence of this organism in the wounds in the patients treated in the disaster is a possible confirmation of this effect It should be pointed out, however, that little is known regarding the absorption of boric acid from burn or granulating surface wounds, and since boric acid has been occasionally reported to give rise to toxic symptoms, it may prove wise after further investigation to omit the boric acid and use plain petrolatum for burns In this way, excessive absorption from large areas would be avoided †

A bland ointment usually gives prompt relief of pain Apparently any oily substance, perhaps because it excludes air from the wound, is comfortable If pain persists, it may be due to too tight a dressing

*Pressure Bandage and Splinting*—The indications for the use of pressure dressings on the extremities and face are not clear to us Pressure dressings on the extremities do prevent the occurrence of massive edema beneath the bandages but may not prevent the loss of plasma In the patients of the Cocoanut Grove disaster, the fluid which seeped out in the burned hands, and which would have formed edema locally, was expressed up the arm and produced massive edema Edema of the burned faces developed despite the pressure dressings Where the scalp was not burned in addition to the face, edema fluid from the face was expressed posteriorly and a generalized edema beneath the bandage was produced Edema fluid was also expressed downwards into the soft tissues of the neck and over the shoulders and upper chest (color section Fig 15 b) (Massive edema of the breasts devel-

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\* The solutions of both tannic acid and the triple dyes recommended for the treatment of burns are strongly acid,  $pH$  2.0 and 2.5, respectively Neutralized solutions were not tried It is possible that the retardation of healing was due to the acidity rather than to anything specifically involved in the tannate or the dyes

† Since this article was written, the urine of 20 of the patients has been analyzed for boric acid A maximum of 2 Gm was excreted in 24 hours in the first two days in the patients with extensive burns Later on when boric acid solution was used to irrigate the granulating wounds, as much as 2.5 Gm were excreted in the urine in 24 hours These levels are far below those of excretion reported in patients having toxic symptoms from boric acid poisoning Since all our patients maintained normal kidney function it is probable that the excretion of boric acid was prompt, toxic levels from absorption were not approached, and the use of boric acid in petrolatum is safe

oped in Case 13) Distended edematous tissues were encountered on performing a tracheotomy where there was no superficial burns of the neck.

It is of course possible that an excessive edema beneath a burn wound may tend often to increase tissue damage. On the other hand we have no evidence that healing was expedited in any of our cases by the use of pressure dressings.

Splinting of an extremity is comfortable and rolled newspapers were incorporated in an outer layer of the pressure dressings. Such splinting decreases lymph flow which may not be of benefit in a sterile burn wound but is in a septic wound. Lymph stasis facilitates the localization of the septic process.

*Administrative Advantages of a Simple Treatment*—From the administrative point of view in handling casualties of a disaster there are many advantages of a simple treatment. The problems of personnel are the most important. In battle or in a civilian disaster there is inevitably a disproportionate number of trained medical personnel to casualties. The simpler the treatment the fewer the trained personnel required to administer it. The treatment used on the patients at this hospital from the Coconut Grove fire can be applied by nurses or orderlies. Physicians present are freed for the administration of plasma for shock and oxygen for anoxia. The operating room and a general anesthetic are dispensed with. The operating room facilities are freed for the care of other injuries. This hospital had seven operating rooms with personnel in preparation for the patients of this fire. When it was obvious that no injuries requiring operative treatment had been sustained the personnel was made available for other work. There is no theoretic reason why burn wounds should have both first aid and definitive treatments. The elimination of the definitive treatment a relic of the tannic acid regimen spares much wasted effort on the part of personnel and discomfort to the patient.

Another advantage of a simple treatment from the administrative point of view is the problem of equipment supplies and their storage. Ideally nothing should be required for the treatment of a burn which is not also useful for some other type of wound. The substances required for the treatment described are useful in other types of injuries. In contrast the paraffin treatment with its heater and spray gun complicates the requirements and rules out this method even though it may well give equally good results. Tannic acid and triple dyes have not been recommended for injuries other than burns. Such considerations of equipment are of paramount importance to the Army and Navy where transportation and storage are problems.

*Medical Advantages of Simplicity*—The treatment of a heat burn should ideally be directed toward the prevention of its complications. Of these the most important are infection and shock. Since infection originates almost entirely from contamination with organisms arriving on the burn surface after the burn has occurred the earlier the wound is protected the less will be the infection. It is clear that the simpler the treatment the sooner it can be applied.

Infection is to be avoided not only because of the shock due to infectious toxemia but also because it delays wound healing and adds to scarring. Indeed, it is probable that the growth of various organisms on burn wounds destroys otherwise viable epithelium and may convert a deep second degree burn into a full-thickness destruction of the epithelium. From the point of view both of survival and early convalescence, infection is most objectionable.

The simpler the treatment, the less manipulation there will be of the patient. From the point of view of aggravation of shock this is important. The rolling of the patient, incident to débridement and washing, not only increases pain but disturbs further the circulatory imbalance impending in shock. Since a simple treatment with minimal requirements of materials will be applied earlier, quicker relief of pain will be obtained, and this too helps to prevent shock.

*Results*—The results of the treatment of the burn surface have been considered by the Staff as gratifying. The second degree burns healed promptly without evidence of infection, and with minimal scarring. Examples are shown in the colored photographs (color section Fig 13 a-e and color section Fig 15 a-e). Of the deep burns, the wounds remained unusually free of active or invasive infection. In his article, Dr Lyons details the course of bacteriologic flora of the wounds and the success of the chemotherapy. Cultures of the burn wounds were not obtained until the time of the first change of dressings so the amount and nature of the contamination present at the time of entry is not known. The subsequent lack of invasive infection was presumably due to the chemotherapy rather than to any unusual cleanliness of the burns on arrival for they were grossly dirty, and there were many chances for fecal and respiratory tract contamination before arrival at the hospital.

Clinical proof that the original surface treatment used, did not lead to infection, and, indeed, on the contrary, was effective in checking the growth of organisms which were present, is shown by the experience with the left hand of Case 2 (color section Fig 14 a-j). Two extensor tendons were exposed on the dorsum of the hand and Dr Cannon elected to graft the hand by burying it in the abdominal wall. Had there been active infection in this wound the tendon would not have survived, the graft would not have become attached, and the abdominal wall wound would have suppurated.

On the fifteenth day after the disaster, the isolation floor was closed. Sixteen patients by this time had been discharged home with lungs free of signs and with surface burns healed. Another patient with a small third degree burn of an ankle had been discharged on the fourteenth day to a Naval Hospital, the second degree burns having healed. (Seven patients had died as the results of the pulmonary complications.)

Of the 15 patients left in the hospital, four were held for residual pulmonary signs, the second degree surface burns of three having healed. (The fourth had no surface burns.) These four were discharged from the seventeenth to the thirty-second day.

The patient with the central nervous system damage from anoxia re-

nained the minor burns having healed by the tenth day. She was discharged on the sixty seventh day.

The remaining ten patients were those with the third degree burns. Dr Cannon describes in his article the wound healing observed in these. The last of these was discharged on the one hundred and forty third day.

COMMENT—The problem of debridement and cleansing cannot be categorically answered for all burns. If a burn surface has been rubbed in dirt more infection may be avoided by débriding and cleansing. In some cases a sluice of water over the surface without debriding may be nearly as effective as any other method. The point to emphasize is that neither debridement nor cleansing are essential to the good care of burns. Good surgical judgment in their care as in that of many other diseases consists of knowing when not to interfere.

The advisability of using pressure dressings on extremities and face has not been settled by the cure of the patients of the Coconut Grove fire at this hospital. The dressings undoubtedly tend to restrict the loss of plasma and therefore the decrease in plasma volume but they do not prevent it particularly in deep burns of the face and scalp when the edema fluid collects in the neck and over the shoulders and chest. A point more important to settle than the effect of pressure on the loss of plasma volume however is whether local edema of the burn tissue is harmful and whether pressure improves the local nutrition. We have obtained no objective evidence on this point.

#### CONCLUSIONS

A treatment for the surface wounds of burn casualties is described and its rationale discussed. It consists of no débridement, no cleansing, a bland ointment with protective dressing and internally administered chemotherapy. It was given extensive trial on the surface burns of the casualties from the Coconut Grove fire treated at the Massachusetts General Hospital and proven eminently satisfactory. Its advantage lies in its simplicity. The available personnel is freed for the care of shock and anoxia yet the surface wounds need not be neglected.

#### REFERENCES

- <sup>1</sup> Davidson, E. C. Tannic Acid in the Treatment of Burns. *Surg. Gynec. & Obst.*, 41: 202, 1925.
- <sup>2</sup> Field, M. E., Drinker, C. K. and White, J. C. Lymph Pressures in Sterile Inflammation. *J. Exper. Med.*, 56: 363, 1932.
- <sup>3</sup> Colebrook, L., and Maxted, W. R. Antisepsis in Midwifery. *J. Obst. Gynec. Brit. Emp.* 40: 966, 1933.
- <sup>4</sup> Jennison, M. W. and Sizer, I. W. The Disappearance of Bacteria Applied to the Human Skin. From Report of Proc. Third Internat. Congress for Microbiology, p. 259, 1939.
- <sup>5</sup> Burtenshaw, J. M. L. The Mechanism of Self Disinfection of the Human Skin and its Appendages. *J. Hygiene*, 42: 184, 1942. *Idem* Mortality of Haemolytic Streptococcus on Skin and on Other Surfaces. *J. Hygiene*, 38: 575, 1938.
- <sup>6</sup> Cannon, B., and Cope, O. Rate of Epithelial Regeneration. *ANNALS OF SURGERY*, 17: 85, 1943.

# PROBLEMS OF INFECTION AND CHEMOTHERAPY\*

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THERE is probably no surgical wound more difficult to evaluate from the point of view of bacterial infection than a deep burn. It is impossible to assess the depth and extent of the fresh burn, and a large number of different types of bacteria are recoverable from the burn surface. During the phase of healing it is frequently impossible to determine to what extent the bacterial contamination has contributed to tissue necrosis. The pathogenicity of certain bacteria may be obvious but with others there may be considerable question. Bacteria which, in pure cultures, are classified as saprophytes may in mixed cultures be pathogens in consequence of synergism. It is usually difficult or impossible to determine the quantitative aspect of the initial contamination or the resistance of the host.

TABLE I  
SULFONAMIDE BLOOD LEVELS DURING THE FIRST TEN DAYS OF TREATMENT

Level mg %	Subsequent		
	Initial	Minimal	Maximal
Less than 2	0	2	0
2—3	2	5	2
3—4	2	6	1
4—5	12	14	1
5—6	6	0	2
6—7	1	0	5
7—8	5	1	8
8—9	2	0	4
9—10	0		2
10—11			3
11—12			0
Total No. of patients	30	28	28

Certain principles of treatment were agreed upon during the period of arrival of the first patients. In general, it was held that

- 1 The prevention and treatment of shock must be the first and immediate consideration

- 2 Further contamination of wounds must be avoided

- 3 Measures to control the established bacterial contamination would be limited to increasing the resistance of the host by chemotherapy, passive immunization and maintenance of nutrition

The rôle of shock in infections cannot be overemphasized. The hemolytic streptococcus is frequently a preferential anaerobe,<sup>1</sup> and one of its lethal toxins is oxygen-sensitive.<sup>2</sup> The importance of shock in gas gangrene, and *vice versa*, was well recognized in World War I. Any factor which retards the circulation and lowers tissue oxygenation invites anaerobic growth of

\* The work described in this paper was done under a contract, recommended by the Committee on Medical Research, between the Office of Scientific Research and Development and the Massachusetts General Hospital

bacteria. Tetanus was a special risk of the thermal trauma from frostbite in the Russian army during the Finnish campaign.<sup>2</sup> On the other hand postoperative shock is often the outward manifestation of hemolytic streptococcus wound infection.<sup>4</sup> Hence it would appear that the prophylaxis of infection is an integral part of the shock problem as it applies to burns. Clinical experience with established infections indicates that an effective blood level of sulfonamide offers the most certain control of systemic infection due to the hemolytic streptococcus. The prompt intravenous administration of a known amount of sulfonamide seemed to be the most direct method of controlling shock due to infection.

TABLE II  
COMPLICATIONS ATTRIBUTED TO SULFONAMIDE THERAPY FOR SEVEN OR MORE DAYS

Complications	No. Patients
Hematuria* only	14
Crystalluria only	3
Hematuria and crystalluria	9
Fever	2
Fever and rash	2
Agranulocytosis	1
Microscopic	

In recording this clinical experience with the victims of the Coconut Grove fire every effort has been made to distinguish between factual data and inference from clinical observations. It seems wise to maintain this distinction here.

#### FACTUAL DATA

*Asepsis and Sterile Precautions*—A strictly aseptic technic was not practical for the initial reception of the patients. All hospital attendants in the receiving ward were masked but it was impossible to mask patients because of the face burns and respiratory difficulties. Clean unsterile gowns

TABLE III  
TEMPERATURE RESPONSE 72 HOURS AFTER STARTING PENICILLIN

Case No.	Diagnosis	W. B. C.	Maximal Temperature Degree Fahr	
			5th Day	9th Day
10	Pulmonary burn	13,400	102	100.5
12	Pulmonary burn and CO poison	13,000	102	101
2	Pulmonary and deep burn	12,600	104.5	100.5
8	P. tracheary and deep burn	27,000	103.5	101.5
11	Pulmonary and deep burn	13,000	104	102.5
13	Pulmonary and deep burn	23,400	103	102
20	Pulmonary and deep burn	17,000	102	103
23	Pulmonary and deep burn	24,000	102	101.5
28	P. tracheary and deep burn	20,000	102.5	101.5
36	Pulmonary and deep burn	13,800	102	101.5
38	Pulmonary and deep burn	26,000	102	102

were donned by volunteer assistants in street clothes but this was not universal. Hospital attendants wore their usual clothing without other covering. Morphine sedation and intravenous fluid therapy were started as soon as possible after arrival.

The rapid transfer of patients from the receiving ward to the ward isolated for their care facilitated supervision of sterile precautions. At



# CHAMP LYONS

In this group of patients the level varied roughly between four and ten mg per cent. No patient developed an abnormally high blood level. The maximal period of treatment was 134 days in one patient.

The complications attributed to sulfonamide therapy in 26 patients receiving drug for seven or more days are listed in Table II.

It was necessary to administer alkali and alter dosage to control hematuria and crystalluria but no patient developed costovertebral tenderness or anuria. The drug had to be omitted in five patients. Two with fever, two

TABLE VII  
CASE 13 DISCHARGED ON 143RD DAY REQUIRED SKIN GRAFTING

Weeks	1st	2nd	3rd	4th	5th	6th	7th	8th
Treatment								
Penicillin								
Sulfadiazine								
Organisms								
Staph, coag +	+	+	+	+	+	+	+	
Staph, coag —	+		+	+				+
Beta strep, group A	+	+						
Beta strep, other								
Alpha strep	+	+		+				
Gamma strep, aer				+		+		
Gamma strep, anaer								
<i>E coli</i> , etc		+						
<i>B pyocyaneus</i>						+		
<i>B proteus</i>			+	+	+	+	+	
<i>Cl welchii</i>				+				
Other Clostridia				+	+	+	+	
Diphtheroids	+	+	+	+	+	+	+	+
<i>B subtilis</i>	+	+	+	+	+	+	+	+

with rash, and one with agranulocytosis. Agranulocytosis was apparent in Case 20 on the twenty-fifth day. Sulfadiazine was promptly omitted. The white blood cell count dropped to 1,000 on the twenty-sixth day, and she was started on pentnucleotide therapy. She had been receiving penicillin since the sixth day and its administration was continued. There was a prompt remission of the agranulocytosis by the thirtieth day, when the white blood cell count had returned to 8,500.

**Tetanus Prophylaxis** Members of the armed forces remaining in the hospital for more than two days received "booster" doses of tetanus toxoid. Civilians were skin-tested with a 1:10 dilution of horse serum the morning after admission, when shock was obviously controlled. No positive reactions were observed and 3,000 units of tetanus antitoxin were given all civilian patients with surface burns, with three exceptions. The exceptions were individuals with severe respiratory difficulty in whom it was desirable to avoid any risk of bronchospasm.

**Penicillin Therapy** Eleven patients were noted on the sixth day to have temperature elevations to 101° F. or higher by rectum. In many of these a real leukocytosis was present. In spite of the clean appearance of the wounds, it was felt that infection was probably present to produce these changes. Coagulase-positive staphylococci had been demonstrated to be the predominant pathogens in the respiratory tract of the patients dying of respiratory obstruction and similar organisms had been recovered from the

# INFECTION AND CHEMOTHERAPY

throats of many of the surviving patients and attendants. The problem discussed with Dr. Chester S. Keefer, who agreed that penicillin might be helpful. A dosage plan of 5,000 units every four hours muscularly was selected. The first concentrations contained 5,000 in 5 cc. of physiologic salt solution but these caused considerable dis-

TABLE VIII  
CASE 28 DISCHARGED 84TH DAY REQUIRED SKIN GRAFTING

Weeks	1st	2nd	3rd	4th	5th	6th	7th
Treatment							
Penicillin							
Sulfadiazine							
Organisms							
Staph. coag. +	+	+	+	+	+	+	+
Staph. coag. -	+	+			+	+	+
Beta strep., group A		+	+		+		+
Beta strep., other							
Alpha strep.	+					+	
Gamma strep., aer.						+	
Gamma strep., anaer.							
E. coli etc.			+	+		+	+
B. pyocyaneus				+		+	+
B. proteus					+		
C. welchii	+						
Other Clostridia							
Diphtheroids	+	+	+	+		+	+
B. subtilis	+	+	+	+	+		+

TABLE IX  
CASE 25 DISCHARGED ON 83RD DAY REQUIRED SKIN GRAFTING

Weeks	1st	2nd	3rd	4th	5th	6th	7th
Treatment							
Penicillin							
Sulfadiazine							
Organisms							
Staph. coag. +	+	+	+	+	+	+	+
Staph. coag. -		+				+	
Beta strep., group A							
Beta strep., other							
Alpha strep.	+						
Gamma strep., aer.					+		
Gamma strep., anaer.							
E. coli etc.	+	+	+				
B. pyocyaneus							
B. proteus				+	+	+	
C. welchii	+	+	+				
Other Clostridia							
Diphtheroids	+	+	+	+	+	+	
B. subtilis	+	+	+	+	+		

when injected into the muscle of the thigh. Much less pain resulted when the 5,000 units of penicillin were dissolved in 1 cc.

Thirteen of the patients received penicillin at some time or other. One patient (Case 16) received the drug unintentionally for one day; a second (Case 29) received the drug during five days of a successful skin grafting late in the period of hospitalization. The remaining eleven received penicillin from the sixth to the fourteenth day or longer. The temperature response in these eleven patients is indicated in Table I.

Accurate appraisal of the efficacy of the penicillin therapy is unfortunately impossible. The dosage employed is now known to have been too

# CHAMP LYONS

in light of subsequent observations by Dr Keefer and ourselves. It was also given in the majority of the patients along with sulfadiazine. No toxic reactions were attributable to the penicillin.

Tables IV through XII summarize the bacteriologic findings in the patients with deep burns requiring skin grafting and treated with penicillin.

Table XIII summarizes the bacteriologic picture of the surface wounds. The initial contaminating flora was a mixture of staphylococci, alpha hemolytic streptococci, diphtheroids and *B subtilis*. With the exception of the alpha hemolytic streptococcus these strains persisted as long as the wounds were unhealed. The group A beta hemolytic streptococcus was present initially in three patients and was subsequently implanted in three more patients. The secondary contamination occurred after the original strict

TABLE X  
CASE 28 DISCHARGED ON 67TH DAY REQUIRED SKIN GRAFTING

Weeks	1st	2nd	3rd	4th	5th	6th	7th	8th	9th
Treatment									
Penicillin									
Sulfadiazine									
Organisms									
Staph, coag +	+	+	+	+	-		+	+	
Staph, coag -	+	+	+	+					
Beta strep, group A									
Beta strep, other			+						
Alpha strep									
Gamma strep, aer	+								
Gamma strep, anaer									
<i>E coli</i> , etc									
<i>B pyocyaneus</i>									
<i>B proteus</i>			+	+	+		+	+	
<i>Cl welchii</i>									
Other Clostridia									
Diphtheroids	+	+	+	+			+	+	
<i>B subtilis</i>	+	+	+	+	+			+	

isolation was relaxed at the beginning of the third week. Anaerobic Clostridia were present initially but tended to disappear. A sufficient number of these anaerobes persisted to warrant immunization to tetanus. The most interesting finding was the distribution of the proteolytic gram-negative bacilli. *E coli* was present initially in a few cases and tended to persist. *B proteus* appeared at the end of the second week as the eschars of deep burns began to separate and was present in all unhealed wounds by the fifth week. *Pseudomonas aeruginosa* (*B pyocyaneus*) was an inconstant contaminant.

## INFERENCE FROM CLINICAL OBSERVATIONS

This program of treatment adequately controlled invasive infection. There was no evidence of cellulitis, lymphangitis, lymphadenitis or bacteremia. Superficial burns healed without suppuration in the usual 10- to 14-day period. The deep burns were all frankly infected and suppurative, but there was no evidence of marginal ulceration of healthy tissue. The outstanding feature of the infection was its limitation to the tissue devitalized by the original thermal injury. Pus formation was associated with the separation of the burn eschar and the presence of proteolytic gram-negative

# INFECTION AND CHEMOTHERAPY

lacilli During this phase of slough local dressings of saline compresses were applied and the process of separation was hastened by bloodless surgical excision of the burn eschar

Fever leukocytosis and positive cultures from the wounds were observed as long as the wounds remained unhealed On the other hand the decision for skin grafting was based more upon the clinical appraisal of the wound

TABLE XI  
CASE 29 DISCHARGED ON 40TH DAY REQUIRED SKIN GRAFTING

Weeks	1st	2nd	3rd	4th	5th
Treatment					
Penicillin					
Sulfadiazine					
Organisms					
Staph. coag. +	+	+	+	+	+
Staph. coag. -	+		+		+
Beta strep., group A					
Beta strep., other					
Alpha strep.					
Gamma strep. aer					+
Gamma strep. anaer					
E. coli, etc.				+	
B. pyocyaneus				+	
B. proteus	+		+	+	+
Cl. welchii					
Other Clostridia					
Diphtheroids	+	+	+	+	
B. subtilis	+	+	+	+	+

TABLE XII  
CASE 36 DISCHARGED ON 58TH DAY REQUIRED SKIN GRAFTING

Weeks	1st	2nd	3rd	4th	5th
Treatment					
Penicillin					
Sulfadiazine					
Organisms					
Staph. coag. +	+	+		+	
Staph. coag. -	+		+		
Beta strep., group A	+	+	+	+	
Beta strep., other					
Alpha strep.	+		+		
Gamma strep., aer					
Gamma strep., anaer					
E. coli, etc.	+	+	+	+	
B. pyocyaneus					
B. proteus	+				+
Cl. welchii					
Other Clostridia	+				
Diphtheroids		+	+	+	
B. subtilis	+	+	+	+	

and the nutritional status of the patient than upon the bacteria present in the wound

In summary it may be stated that the method of treatment confined the bacterial infection to the devitalized tissue and protected the living cells from invasive infection or marked local necrosis.

Discussion.—The inability to accurately identify the depth of thermal injury in the immediate posttraumatic period of observation is a serious handicap in the final evaluation of any method of treatment. It may be impossible to distinguish between a deep burn and an infected superficial

## CHAMP LYONS

burn by an examination of the wound prior to skin grafting. Our experience indicates that a superficial burn heals kindly with any method of treatment that prevents destructive hemolytic streptococcal infection. It is equally true that no method of treatment has arrested the growth of bacteria in the deeply burned tissues. The treatment adopted in this group of cases was based upon the premise that the preservation of the vitality of the undamaged cells was a realistic objective in the treatment of burns. It is believed that reasonable success attended the use of a nonadherent occlusive local dressing and systemic supportive and antibacterial therapy. The failure to sterilize the devitalized burned tissue by such a method was not unexpected.

TABLE XIII  
SUMMARY OF BACTERIOLOGIC FINDINGS

Weeks	1st	2nd	3rd	4th	5th	6th	7th	8th	9th
No of cultures	24	17	10	9	9	5	6	4	1
Organisms									
Staph, coag +	20	16	9	8	7	5	5	3	1
Staph, coag —	15	10	6	3	3	3	4	1	
Beta strep, group A	3	4	3	2	1		1		
Beta strep, other	1		3						1
Alpha strep	15	5	1	2		1			
Gamma strep, aer	1	1	1	1	2	2			
Gamma strep, anaer		1			1				
<i>E coli</i> , etc	5	4	4	2			1		
<i>B pyocyaneus</i>	1	1	3	2		3	1	1	
<i>B proteus</i>	2	1	4	6	9	3	5	2	
<i>Cl welchii</i>	6	2	1	1					
Other Clostridia	2			1	1	1	1		
Diphtheroids	17	15	10	8	5	5	6	3	1
<i>B subtilis</i>	21	16	9	9	6	1	4	2	

Indeed, if it is ever possible to sterilize such tissue, the removal of the eschar may become a major problem.

The observation of this group of patients with controlled and limited infection has thrown into sharp contrast the derangement of bodily homeostasis incident to the traumatic insult of a major burn. This damage plus the metabolic burden of infection depletes the nutritional reserves and retards the convalescence of the patient. Resistance to infection and tissue repair are intimately correlated with recovery from this phase of negative balance.

### CONCLUSIONS

The problem of preventing infection in burns has three components: Prevention of further contamination of wounds; effective antibacterial therapy for established contamination, and supportive measures to maintain the reparative and convalescent capacities of the patient.

### REFERENCES

- 1 Jones, M, Holmes, L. F, and Hale, W. M. Relative Efficiency of Aerobic and Anaerobic Methods in the Isolation of *Beta* Hemolytic Streptococci. *J Bact*, 42, 138, July, 1941.
- 2 Todd, E. W. Lethal Toxins of Hemolytic Streptococci and their Antibodies. *Brit J Exp Path*, 19, 367, December, 1938.
- 3 Acutin, M. N. *Trans Kubyshev Milit Med Acad Red Army*, 2, 3, 1940.
- 4 Stewart, J. D. Postoperative Shock Due to Hemolytic Streptococcus Wound Infection. *Surgery*, 9, 204, February, 1941.

# PROCEDURES IN REHABILITATION OF THE SEVERELY BURNED

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FOURTEEN of the 39 patients admitted on the night of the disaster remained in the hospital after the special casualty ward was discontinued on December 13. Ten of these patients had third degree burns of sufficient extent to require further treatment. Only one of the ten was hospitalized for as long as 18 weeks. The average stay of the others was ten weeks; the minimum was three and one half weeks and the maximum fourteen and one-half. During this time, one or more skin grafting operations have been performed on nine of the ten patients. The tenth patient had a small area of deep burn on the back which rapidly healed from the margins without grafting.

*Distribution of Third Degree Burns*—The skin on the dorsum of the hand was the surface most frequently destroyed by third degree burn (fifteen hands in nine cases [Cases 2, 8, 11, 13, 20, 23, 28, 29 and 36] with both hands in six). The arms were the next most frequently involved surfaces (ten arms in six cases [Cases 2, 8, 11, 13, 20 and 28] both being involved in four). The back was deeply burned in six patients (Cases 1, 8, 11, 13, 20 and 28) and the scalp and forehead in four (Cases 2, 8, 13 and 29). In three cases (Cases 2, 11 and 13) the legs were deeply burned and in one of these (Case 13) the third degree burns extended almost completely around both lower legs and the lower third of both thighs. Scattered areas of the neck were deeply burned in Cases 8, 13, 20, 23 and 36.

Because of the similarity in distribution of most of these burns it is interesting to speculate about the position taken by the victims as the fire spread rapidly across the ceiling. The hands were held protectively over the face, and the faces were deeply burned in only two cases. In one of these (Case 8) there were three vertical linear areas of third degree burn on the forehead. It appears that the forehead was burned between the separated protecting fingers. The extensor surfaces of the forearm and the lateral surfaces of the upper arm and shoulder were deeply burned in two cases (Cases 8 and 20) both women in evening dresses who exposed these surfaces as the hands were held over the face for protection. Both of these women were burned on the back and in both the pattern of the underwear is evident (color section Fig. 12 a). It appears that the victims leaned forward and turned away from the most intense heat and were thus severely burned on the back as well as on the arms and hands.

*Treatment of the Local Wound*—The primary occlusive dressing of a fine mesh gauze lightly impregnated with boric acid ointment\* and applied with firm pressure has already been described (color section Fig. 15 a). Similar

\* Boric acid ointment, 10 per cent, in petrolatum.

dressings were used after the first were changed between the fifth and tenth days. By the tenth to twelfth day the destroyed skin had begun to separate and there was suppuration about the margins with pus escaping through openings in the slough. No cellulitis or lymphangitis indicative of invasive infection was apparent. In order to insure better drainage of the wounds, constant wet dressings, with a single layer of fine-mesh gauze against the granulations, were applied at this time. These were kept moist by instilling 2 per cent boric acid solution through Dakin's tubes incorporated in the dressing. They were changed every second or third day. An alternation of wet and boric ointment dressings was tried, but because of the discomfort of the latter, especially on the hands, the wet dressings were continued until the time of grafting. The Bunyan envelope was used for several days in four patients (Cases 2, 11, 28 and 29) who had burns of the hands and forearms. The patients found them uncomfortable. The common complaint was of the humidity of the atmosphere when the bag was filled with oxygen and the burning pain when filled with normal saline. The absence of support for the hand and discomfort on contact of the hand with the envelope were also commented upon. Strands of slough and exudate were suspended in the water but did not separate. However, no hypochlorite was used, as Bunyan recommends.

*Separation of Slough*—The average time before complete separation of the slough was twenty-five days. The maximum was thirty-six days, and the minimum sixteen days. This average interval is longer than one normally expects the process to take. There appears to be no correlation between the patient's general condition, the duration of chemotherapy, the extent of the burns, and this delayed separation. The relative freedom of the wounds from invasive infection may be of significance, as there was little pus beneath the slough when it was finally removed. Scattered islands of viable skin, 1 to 2 cm in diameter, remained on the backs of some of the hands and arms after separation of the slough (color section Fig 16 d). These had not been destroyed secondarily by infection.

*Splinting of Hands*—An effort was made to hold the hands in the position of function but this could not be continued because of the pain. Flexing of the fingers put the open wounds on the dorsum under tension, and the patients could not tolerate it. Flexion also caused blanching of the granulations or skin over the metacarpophalangeal and interphalangeal joints, which increased the danger of further necrosis of tissue and possible exposure of the extensor tendons and the joints. Therefore, the fingers were allowed to remain in extension and the wrist in a neutral or slightly dorsiflexed position. This position was maintained until operation, at which time the hand and fingers were splinted in a more nearly normal functional position.

*Preparation for Grafting*—After the special casualty ward was discontinued all dressings were done in an operating room set aside for these cases. The use of wet dressings applied with moderate pressure was continued after the slough had separated. The exposure of the raw surface,

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## COLOR SECTION

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*Containing 16 figures (49 subjects) in color to which reference has been made in several papers throughout the Symposium. The figures are numbered in this section in their own series 1-16 a, b, c, etc. Corresponding text citations specify their appearance in this group.*



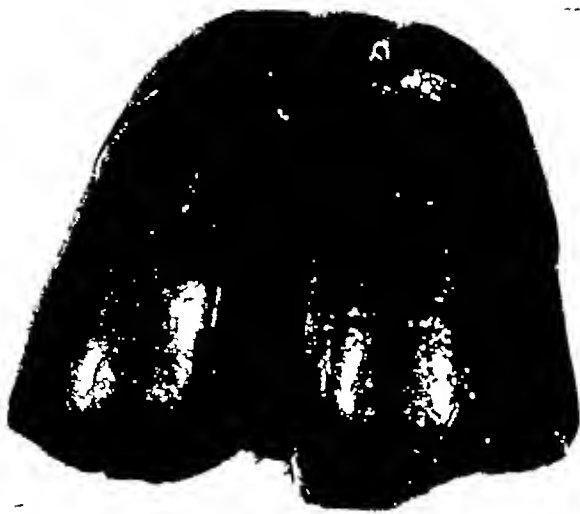


FIG 1—Lungs of case dead on arrival The arresting feature is the bright pink color, attributable to carbon monoxide. An occasional subpleural hemorrhage can be seen. They are uniformly distended.



FIG 2—Lungs of Case 25. They are voluminous due to extensive anatomic emphysema. The posterior and central portions are deeply congested where the tissue can be seen through the dense anthracosis. Around the margins of all lobes is a light-colored zone two to four lobules wide in which air is trapped by the diffuse bronchostenosis.



FIG. 6—A segment of the bronchial tree from Case 25. All mucous membranes are intensely red. In the secondary bronchi membranous bronchitis begins in spotty fashion. In the smaller bronchi the membrane becomes continuous and frequently occlusive. The pulmonary parenchyma is deeply congested and focally anthracotic.

FIG. 7—Bronchial tree from Case 27. The mucous membranes are hemorrhagic and membrane formation is present though less pronounced than in Case 25. Clots protrude from the severed vessels which are, in part, antemortem. The parenchyma shows alteration of aeration and atelectasis.

Fig. 6



Fig. 7



FIG 8 —Posterior view of the lungs of Case 27. The basal portions of both lower lobes are massively atelectatic, dark red in color, depressed in contrast to the aerated parenchyma. Narrow prolongations of the atelectatic zone extend upward to the apices of the lobes. Another atelectatic zone can be seen on the lower margin of the left upper lobe.

FIG 9 —Sagittal section of lung of Case 27. Membrane formation is visible in the secondary bronchi. A wide zone of dark, depressed, atelectatic tissue occupies the central part of the lower lobe. Aerated tissue is present above and below the area. The portion of upper lobe visible is all aerated.

FIG. 8



FIG. 9



FIG 10—View of the temporary operating room during the initial change of dressing on Case 20 on the seventh day. Surgeon is reapplying boric strips to face. Patient has had a tracheotomy. The dressings on the deep burns of the arms and hands are typical. (Note bacteriologist in right background and the photographer.)

FIG 11 a and b Case 13—Appearance of circular burns of legs on tenth day. This was the most severely burned patient who survived. She also had deep burns of the hands and arms, scalp, forehead, and back, total extent of second and third degree was 56 per cent. Note the dry slough above the right knee and the moist slough immediately adjacent on the lateral aspect of the thigh.

FIG 11 e. Case 13—Legs on 71st day. A striking change has occurred, there has been a rapid advance of epithelium from the margins and the granulation tissue now has a healthy appearance. This change is attributable to the improvement in the patient's general condition from intravenous feeding and repeated transfusions.

FIG 11 f Case 13—Legs on 81st day, tenth day after grafting. There is a complete take of the grafts. The small unhealed areas were not covered at the operation.

FIG 11 g Case 13—Legs on 91st day. Healing is practically complete.

FIG 11 c Case 13—Legs on the 33rd day after all the slough has separated. The surface is pale and the underlying fat is visible. No granulation tissue has appeared.

FIG 12 a Case 8—Deep burns of the back at the time of the first change of dressing on the fifth day. The outline left by the clothing is visible. Note the dressings covering the deep burns of the arms. This patient also had second and third degree burns of the head, neck, arms, hands and legs, total extent 20.1 per cent.

FIG 11 d Case 13—Legs on 64th day. A thin layer of granulation tissue has developed. There is wasting of both legs resulting from the debilitating effect of chronic sepsis in the open wounds. Contracture of right tendo achillis is present.

FIG 12 b Case 8—Back on the tenth day. The slough has begun to dry. There is exudate about the margins but minimal evidence of inflammation in the adjacent skin.

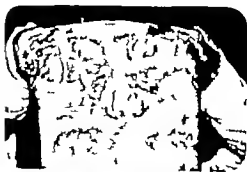
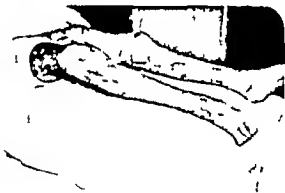




FIG 14 a Case 2—The left hand at the time of the first change of dressing on the fifth day. The outlines of the third degree burn are already apparent. The damaged superficial skin of the fingers and thumb has not been removed. There is slight edema of the hand.

FIG 14 b Case 2—Left hand on ninth day. The slough has begun to separate. Although bacteria have been recovered on culture, there is minimal infection.

FIG 14 c Case 2—Left hand on 13th day. More slough has separated.

FIG 14 d Case 2—Left hand on 19th day. The slough on the wrist was removed mechanically, that on the dorsum of the hand is still adherent. The surrounding skin is uninfected.

FIG 14 e Case 2—Left hand on 26th day. The slough has been removed. The extensor tendons of the index and middle fingers are exposed over the metacarpophalangeal joints. Exudate is still adherent to the granulations.

FIG 14 f Case 2—Left hand on 28th day. Absence of invasive infection permits insertion of hand into abdominal wall pocket in an effort to preserve the exposed tendons. Note the decrease in exudate overlying the granulations in past two days.

FIG 14 g Case 2—Left hand on 28th day, is in the pocket and the raw surface on the wrist has been covered with split graft.

FIG 14 h Case 2—Left hand in pocket on 33rd day, five days after insertion. Absence of virulent infection of hand is demonstrated by paucity of inflammation of pocket walls. There is a narrow zone of cellulitis about the opening through which the index finger emerges.

FIG 14 i Case 2—Left hand on 48th day, detached 20 days after insertion. The color of the flap is good and the edges have been sutured in place. (The nail polish has been present since the night of the fire.)

FIG 14 j Case 2—Appearance of hands at four months. The tendons have been preserved and function is returning. The fullness of the flap is diminishing but some may have to be excised later.

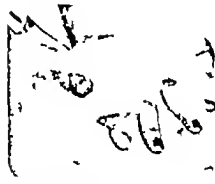
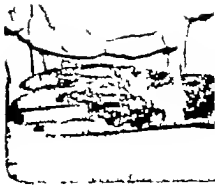


FIG 15 a Case 29—Second degree burns of face and ears and third degree of scalp covered by the primary occlusive dressing which was applied on the night of admission. Only the mouth and nares are exposed but patient can breathe comfortably. Patient also had second and third degree burns of hands and arms, total extent 12.5 per cent.

FIG 15 b Case 29—View of the neck and chest on the third day showing the massive subcutaneous edema in these unburned areas. This edema fluid was expressed from the burns of the face and scalp by the pressure dressing and gravitated downward.

FIG 15 c Case 29—At the time of the first change of head dressing on the seventh day. There is edema of the face in spite of the pressure dressing. The remnants of destroyed skin and dry serum are still present and uninfected.

FIG 15 d Case 29—Face on ninth day. The edema has diminished. There is still weeping from the skin and crusts have reformed. Some skin debris is still present.

FIG 15 e Case 29—Final view of face on the 55th day showing the absence of scarring and return to normal contours. Scalp healed without grafting. (hands and arms were grafted).

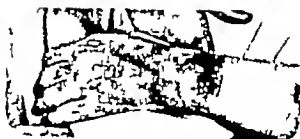
FIG 16 a Case 28—View of the left hand and arm at the time of the first change of dressing on the fifth day. Note the unruptured blebs on the arm and the remnants of destroyed skin on the wrist and fingers. There is no evidence of infection in these wounds. The fluid removed from the bleb is sterile and the sulfadiazine level in this fluid is 6.7 mg per cent (that of the blood 6.6 mg per cent). Patient also had second degree burns of face, neck, back, right hand and arm, third degree of back, total extent 24.5 per cent.

FIG 16 b Case 28—Left hand and forearm on 19th day. Slough is still in place. The skin immediately adjacent is normal in appearance without evidence of infection.

FIG 16 c Case 28—Left hand and forearm on 30th day. Most of the slough has separated. The granulations are edematous. Infection is minimal.

FIG 16 d Case 28—Appearance of the hand and forearm on the 51st day, the day of grafting. Note the healthy appearance of the granulations and the islands of viable skin in the center of the raw surface on the back of the hand.

FIG 16 e Case 28—Final view of the hands at three and one-half months.





especially of the hands was painful and in some cases nitrous oxide oxygen anesthesia was given to help the patient's morale by eliminating the pain and to reduce the time taken for the procedure. The wet dressings acted as a keratinizing stimulus particularly on the areas of second degree burn adjacent to the granulations and on the advancing marginal epithelial surfaces. This debris did not develop when an ointment dressing was applied. The debris which became macerated grew staphylococci and saprophytes on culture. At each change of dressing the surfaces were carefully cleaned with soap and water to reduce as much as possible this source of contamination of the wounds. The persistence of infection and edema of the granulations after the slough had separated may be attributed in part to this maceration. In most of the cases general chemotherapy had been discontinued before the time of grafting. Oral sulfadiazine was given to Cases 8 and 13 both pre and postoperatively and to Case 29 for the first operation. Penicillin was being given intramuscularly to Cases 2, 20, 29 and 36 at the time of their first operations but was discontinued a few days later. All other operative procedures were undertaken without general chemotherapy. Local sulfanilamide powder in controlled doses was dusted on the raw surfaces preoperatively in several cases. The edema subsided and the drainage decreased with this treatment more rapidly than with the wet dressings alone. There were no losses of the graft following the local use of sulfanilamide.

All patients were given a high protein and high vitamin diet throughout the hospital stay. In Case 13 because of inadequate mouth intake it was necessary to feed by stomach tube with supplementary daily intravenous amogen glucose and vitamins. Anemia was controlled by repeated whole blood transfusions. A total of twenty five were given. Two weeks after this intravenous therapy was started the patient's general condition and the condition of the local wounds had improved so much (color section Fig. 11 c) that both legs were grafted. The grafts took completely.

#### OPERATIVE PROCEDURES

*Anesthesia*—Ether anesthesia was used in 13 of the 21 operations. Spinal anesthesia was administered for all operations on Case 2 and was supplemented with sodium pentothal at the first operation and brachial block at the third. Local infiltration was used in Case 29 and spinal in Case 36 when cutting the grafts. No anesthesia was used when applying the grafts to the hands in these two cases. The other operation upon Case 29 was performed with sodium pentothal anesthesia and one of those on Case 13 with spinal. There were no complications from anesthesia in any of the patients who had lung damage.

*Grafting*—Split thickness grafts\* were used for covering the raw surfaces with the exception of the back of the hand of Case 2 which was covered by a direct abdominal flap (color section Fig. 14 a-j). Undamaged extensor

\* The donor areas of the grafts were dressed with a fine mesh gauze lightly impregnated with scarlet red ointment. The overlying gauze sponges were held in

tendons of the index and middle fingers of this hand were exposed over the metacarpophalangeal joints for a distance of 3 to 4 cm. The granulating surface extended from the wrist to the proximal phalanges of the two fingers and involved most of the back of the hand. In order to save these tendons from destruction by infection and to insure normal function in the future, a flap

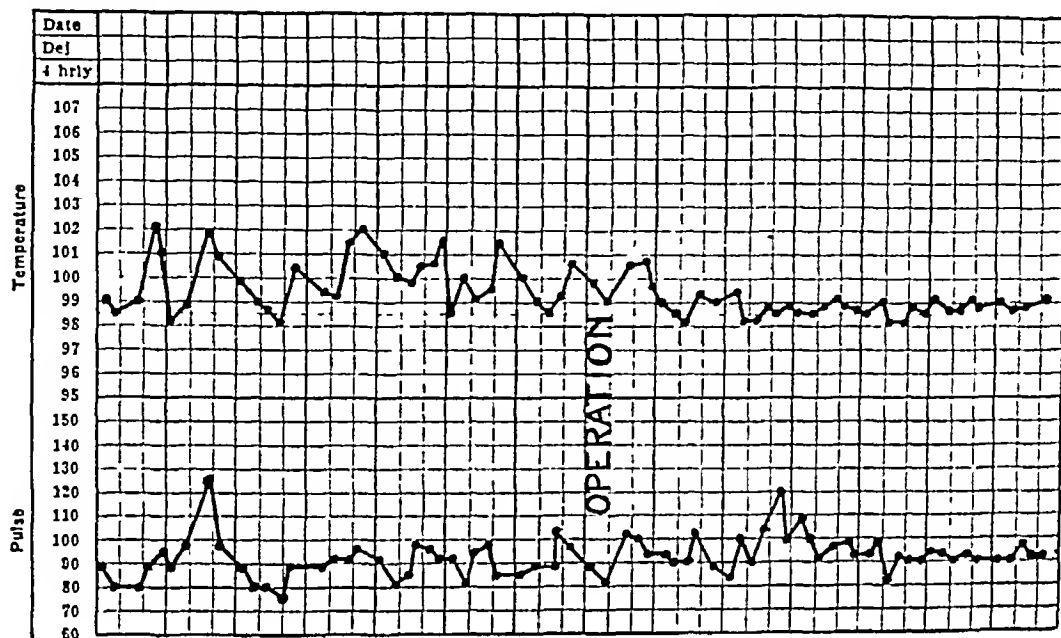


FIG. 41.—Chart of Case 8 at the time of operation on the thirty-ninth day. The preoperative temperature and pulse curves resemble those of all the patients who had large, granulating, surface wounds. Note the prompt descent of the curves to normal after the raw surfaces were covered with skin grafts.

was used instead of a free graft. The operation was performed on the twenty-eighth day. There was little surface infection and culture grew only coagulase-positive staphylococcus and *B. proteus*. The raw surface was gently cleansed with soap and water, but the granulations were not disturbed. A pocket was made in the abdominal wall with a large opening laterally, through which the hand was inserted, and with smaller openings, through which the thumb and fingers emerged. A small raw surface just above the wrist was covered by a split-graft at the same time. The arm was immobilized by adhesive strapping. One border of the flap was severed on the fourteenth postoperative day, and the hand detached on the twentieth day. Three weeks later the defect on the abdominal wall was grafted.

A total of 17 grafting operations were performed on the other eight patients (Fig. 41). The first graft was applied on the twenty-third day to the back of the hand of Case 29, and the last at four months to several small areas on Case 13. Case 36 was grafted on the twenty-fourth day. Six operations were performed during the sixth week (Cases 8, 11, 20, 23, 29 and 36). Two were done in the eighth (Cases 23 and 28). One was performed in the ninth (Case 13). Two were performed in the tenth

place with cotton or ace bandage. When the grafts were taken from the thigh the whole leg was wrapped in an encircling ace bandage. All dressings were removed between the twelfth and fourteenth day unless small unhealed areas were discovered.

## REHABILITATION OF BURN CASES

(Cases 20 and 23) Two were performed in the thirteenth (Cases 11 and 13) And subsequently two minor procedures have been undertaken on Case 13 \*

At operation the raw surfaces were prepared with soap and water. The granulations and the narrow zone of skin that had grown in about the margin were shaved off in all cases except on the hands of Cases 29 and 36. There was a moderate loss of blood in this procedure, but the graft acted as an efficient hemostatic agent and a smooth firm base for it was secured. Most of the grafts were sutured accurately in place but where possible a carefully applied dressing gave adequate anchorage and sutures were not used. The grafts were perforated only if oozing persisted beneath them.

All postoperative dressings were applied wet and kept wet by irrigation through Dakin's tubes. A single layer of fine mesh gauze was used against the graft and the overlying dressing consisted of sponges and a thick layer of mechanic's waste held firmly in place with cotton bandages. Sterile, well padded wood splints were used to immobilize the hands and arms after operation. A bulky dressing was sufficient for fixing the ankles and feet but padded wood splints were used at the knees. The first dressing was done on the fifth or sixth day after operation. Boric ointment gauze was applied in all cases. Subsequent dressings were done on alternate days as long as was necessary.

All the grafts were successful except those on Cases 23 and 36. Both hands were grafted in each of these patients. Operation upon Case 23 was performed on the thirty seventh day and on Case 36 on the twenty fourth day. On the second postoperative day Case 23 had a plasma protein level of 5.5 mg per cent and a hematocrit of 24 per cent, for which two 500 cc. transfusions were given. Cultures at the time of grafting from the left hand showed *Staphylococcus albus* coagulase negative and *B. proteus*. From the right hand was grown hemolytic *Staphylococcus aureus* coagulase positive *beta* hemolytic streptococci *B. proteus* and diphtheroids. Five days before operation in Case 36 the plasma protein was 8.1 per cent and the hematocrit 44 per cent. Cultures of the hands at operation grew hemolytic *Staphylococcus aureus* coagulase positive *beta* hemolytic streptococcus and several saprophytes. Case 23 is the only one showing a significant variation from normal plasma protein and hematocrit at the time of operation and these patients are the only ones having a positive culture of *beta* hemolytic streptococcus from the granulating surfaces. Case 23 was operated upon twice at biweekly intervals before the hands were completely healed. Sulfanilamide powder was used locally before the second of these operations. Case 36 was grafted again 11 days after his first operation.

\* Case 13 received a sternal transfusion on the sixth day. There was extravasation of the blood into the tissues of the chest wall and the breasts. Subsequently localized tenderness, heat, and a bronzed discoloration of the overlying skin developed. A large abscess was drained on the twelfth day. Twenty-one days later a revision of the wound was necessary to give better drainage. The resulting defect, which measured about 12 cm. in diameter was later grafted.



## PROGRESS AND RESULTS

The burns of the extremities especially the hands, were the first surface-grafted for reasons of comfort and early restoration of function. The relatively small areas of third degree burn on the faces were grafted as early as possible for cosmetic reasons.

*Fingers*—On many of the fingers there was a narrow zone of third degree burn on the dorsal surface. Healing occurred rapidly in the two distal phalanges but grafts were necessary for covering the proximal phalanx in fingers of Cases 8, 11, 20, 23, 28 and 36. The extensor tendon of the index and little fingers of the left hand were exposed over the proximal interphalangeal joints in Case 8. Healing is complete but there is absence of full extension at these joints. In Case 20 the proximal interphalangeal joint of the left index finger was exposed and has remained open. There is adequate soft tissue drainage, and ultimately sequestration of bone and destruction of the joint is anticipated. For cosmetic reasons the finger is splinted in extension. Case 13 had these same joints exposed in all four fingers. The fingers have been held in extension with a banjo-splint, and healing has taken place without sequestration. The four fingers of the right hand were so badly burned that only the palmar skin remained viable. Amputation was performed distal to the metacarpophalangeal joints on the thirty-third day. This patient also had a localized deep burn over the lateral aspect of the thenar eminence, with destruction of the underlying muscle and exposure of the metacarpal bone. Spontaneous healing took place following débridement and free drainage.

There has been minimal thickening of the skin on the fingers and no true keloiding in any of the cases.

*Hands*—One or both hands of Cases 8, 11, 13, 20, 23, 28, 29 and 36 were grafted. Most, or all, of the dorsal surface of these hands had to be covered (color section Fig. 16 a-e). In a few of the cases epithelium from early spontaneous healing was not excised at operation and there is now a poor bearing surface easily broken by trauma. In Case 2, who had the direct abdominal flap, the tendons were salvaged, and there is normal extensor function. A thick layer of abdominal fat is present in the flap giving a bulky appearance. This excess fat will be excised at a later date for cosmetic reasons (color section Fig. 14 a-j).

There has been some thickening of the scars at the margins of the grafts, and true keloids have developed on the backs of the hands of Cases 21 and 36. These were the two patients in whom there was loss of the grafts and in whom secondary operations were performed. Because of the scarring the back of the hand is tight and flexion of the fingers and hand tenses the skin of the forearm. Further corrective procedures will have to be undertaken in these cases.

*Arms*—The arms of Cases 8, 11, 20, and 28 were all grafted successfully. Good covering was obtained and there has been good recovery of function without contracture. No keloid has developed.

*Back*—Although the backs of Cases 1 8 11 13 20 and 28 had third degree burns of varied extent only that of Case 11 was grafted. After the slough was removed healthy granulations appeared and spontaneous epithelization occurred quite rapidly. In the meantime all except Case 1 were being treated for deep burns of the extremities. During this period the patients lay on their backs with very little discomfort.

In the central areas which were the last to heal there is keloid developing. This change has not appeared in the marginal surfaces of spontaneous healing which lie nearer the normal skin. No keloiding has occurred in the areas grafted on Case 11. Only Cases 8 11 and 28 have complained of itching of the back but these had extensive burns (color section Fig. 12 a-f).

*Forehead and Scalp*—Case 8 had a third degree burn of the forehead which was grafted. Cases 2 and 29 had burns of the scalp which were small and healed without grafting. Case 13 had a third degree burn of all the forehead and right temple which extended posteriorly in the left parietal region to an area about 12 cm. in diameter over the occiput. In the center of both the forehead and the occiput bare bone was exposed. A 4 cm. disk of outer table of posterior skull has separated spontaneously but a small area of bone is still exposed on the forehead. All of the granulating surfaces have been grafted successfully. No keloiding has developed in these areas.

*Ears*—In Case 13 there was a deep burn of the ear with exposure of cartilage at the rim of the helix. Healing took place when adequate soft tissue drainage was established and minimal loss of cartilage occurred.

*Neck*—Case 13 had a linear third degree burn of the left side of the neck which healed without grafting but there is a moderate contracture. In Cases 8 20 23 and 36 there were narrow bands of deep burn which caused no contracture but which have begun to keloid.

*Legs*—Of the three patients (Cases 2 11 and 13) who had deep burns of the legs only two (Cases 11 and 13) were grafted. They are now healed. Case 13 had extensive raw surfaces which were grafted at four months; there are no contractures but a moderate shortening of the right Achilles tendon has developed. no keloid has appeared (color section Fig. 11 a-g).

#### COMMENT

The protection of all tissue cells not destroyed by the heat is the ultimate purpose of any method useful in the local treatment of burns. Because infection is a destructive process and because destructive organisms grow in traumatized tissue it has been common practice regardless of the surface agent applied to debride all burns at the time of the definitive treatment. The evaluation of the unorthodox method employed in this group of patients depends on determining whether the healing time was prolonged because the burns were neither débrided nor cleansed before the first dressings were applied.

It is well known that uninfected second degree burned surfaces usually heal within two or three weeks. The same interval of healing was ob-

served in this group of patients. Examination of the wounds at the time of the first and subsequent dressings revealed a clean surface with no purulent exudate, cellulitis, or acute tenderness. The destroyed epithelial débris was dry and the contents of the intact blebs were sterile. Therefore, it is apparent that the failure to débride the wounds initially did not delay the healing of the second degree burns in any measurable degree.

In the smaller group of patients who had both second and third degree burns, the injured surfaces remained clean during the first two weeks, with minimal exudate and no cellulitis or acute tenderness. By the end of two weeks the areas of second degree burns were epithelized and uninfected, while in the immediately adjacent areas of third degree burn, slough was separating (color section Fig 16 b). *Satphylococci* and the saprophytes grew in the slough but did not invade the living tissues. When removed, only a thin exudate was found beneath the slough with no abscesses. In the hand of Case 2 exposed tendons were found. These remained viable in the granulating wound and were salvaged by covering them with an abdominal flap on the 28th day (color section Fig 14 a-j). It is apparent from these observations that the wounds were free from invasive infection and that there was no measurable delay in carrying out the reparative procedures.

It seems reasonable to conclude from these observations, both on the second and third degree burns, that débridement and cleansing need not be part of the treatment. Tissue resistance and general chemotherapy can be relied upon to prevent invasive infection. However, for success, an ointment gauze should be used to allow serous drainage into the overlying dressing. The surface must not be sealed by an impervious membrane or eschar. Precautions should be taken to reduce contamination by avoiding careless exposure of the wounds at the primary dressing and too frequent subsequent changes. For this reason and because all wounds in these cases remained so free from infection, the first change of dressing should probably be postponed for 12 to 14 days.

The importance of the general nutritional state in the spontaneous healing of burned surfaces and the preparation of these surfaces for successful skin grafting cannot be overemphasized. Only by early skin grafting can large burned areas be covered and the development of contractures be avoided.

## A NOTE ON PHYSICAL THERAPY

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SIX PATIENTS who received severe burns to the dorsum of the hands and wrists were referred to the Physical Therapy Department either while in the hospital or at the time of discharge to be treated as out patients (Cases 2 8 23 28 29 and 36). In all cases surface healing was complete before beginning treatment. The first patient (Case 29) was referred to this department 51 days after the fire so that the maximum period of physical therapy at the time of writing was 70 days three to five treatments being given each week. There are of course no end results to be reported at this time for continued improvement is anticipated for the next six to twelve months or longer.

### METHODS OF TREATMENT

As these patients were splinted in extension the aim of physical therapy was to restore flexion in the digital joints and wrists also if involved. In the one case in which a direct abdominal flap was undertaken (Case 2) there was the additional problem of mobilizing the shoulder which had developed an adduction internal rotation contracture from the fixation required for this type of transplant.

During the first few weeks of treatment the grafts and newly epithelized areas were quite thin and sensitive to very minimal trauma whether mechanical or due to heat. Because of this a layer of lanolin was gently applied over the new epithelium. The whirlpool bath at a temperature of approximately 104° F. was then used for a period of 15 to 20 minutes. In some cases the initial bath temperature was slightly lower later being increased as tolerance developed. While still in the bath the patients were instructed to start gentle slow steady active flexion of the fingers and wrists up to the point of discomfort. When sufficient flexion allowed the patients were given rubber sponges to grasp as a form of underwater exercise. After several weeks of the whirlpool bath when the epidermis was stronger and thicker the melted paraffin wax bath was substituted for it. The temperature in these baths was maintained at 126° F. and accordingly produced much greater and longer lasting hyperemia and increased relaxation of tense structures. In addition a thin film of oil remained after removal of the wax coating which was an excellent lubricant for massage.

Following this thermotherapy massage of a special type was given. This consisted of gentle circular and longitudinal friction at the junction of the normal and involved areas. The friction was not between the technician's fingers and the skin but between the skin and subcutaneous layers and the underlying bone tendons and muscles. By this method some loosening of

served in this group of patients. Examination of the wounds at the time of the first and subsequent dressings revealed a clean surface with no purulent exudate, cellulitis, or acute tenderness. The destroyed epithelial débris was dry and the contents of the intact blebs were sterile. Therefore, it is apparent that the failure to débride the wounds initially did not delay the healing of the second degree burns in any measurable degree.

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scar could be obtained without traumatizing the new skin. Massage was also given in such a way as to pull the normal skin in the direction of the knuckles in order to relax tension which was maximal at that point. Simultaneously with this stretching of normal skin the patient was instructed to actively flex the fingers as much as possible. Occasionally gentle passive stretching of the firmer bands of scar was done with one hand while massaging with the other. The intensity of this passive procedure, however, was very carefully regulated to avoid traumatizing the new skin.

Exercises followed massage and for the most part were active guided voluntary motions. Emphasis was placed on slow steady stretching to the point of discomfort and degree of skin tension thought advisable as judged by the blanching over the metacarpophalangeal and interphalangeal joints. In addition to intensity, the frequency of repetition of the exercises had to be regulated, both during treatments and at home. It was soon found that excessive movements lead to small blister formation in some patients and avoidance of this was an additional guide to dosage.

An important part of the therapeutic exercise program was teaching relaxation. These patients unconsciously had a tendency to hold the fingers in complete extension and protected the hands from all possible contacts. To overcome this habitual attitude which delayed return of function, they were constantly reminded to make use of their hands in a natural way for the numerous and frequently repeated movements usually pertaining to eating, dressing, gesticulating, *etc*. As the hand is such an intricate mechanical mechanism, actual use in tasks of appropriate degree of difficulty was further advised to increase function, rather than relying on set exercises alone. The occupational therapists were helpful on this score in suggesting and supervising practice work such as typewriting, knitting and light carpentry. Some of the patients took this lead as an incentive to carry on increasingly difficult manual maneuvers on their own initiative, such as developing and printing photographs and playing jackstones.

#### RESULTS

No true end-results are warranted at this date as the physical therapy for these patients has really just begun. It is possible, however, to demonstrate the rate of improvement in function as progress has been recorded by double exposure action photographs.

Case 36 was one of the first to receive physical therapy. Treatments started the sixty-second day, at which time there was only a jog of motion in the finger joints. Action photographs show approximately 50 per cent of normal joint motion 18 days after the first treatment (Fig 42 A) and nearly complete flexion 35 days later after a total of 30 treatments (Fig 42 B).

Cases 2, 8, 23, 28 and 29 had the same type of physical therapy and have made comparable progress with the exceptions noted below.

#### COMMENT

The most striking feature, so far concerning return of function following

A



B

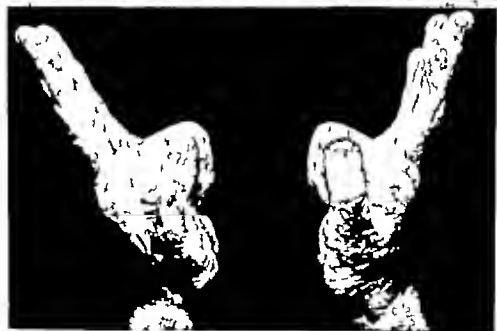


FIG. 43.—Case 36. Double exposure photographs to show maximum extension and flexion of the fingers. (A) February 5; (B) March 12. Thirty physical therapy treatments given starting January 29, 1943.



physical therapy, was the slowness and difficulty in regaining flexion of the metacarpophalangeal joints in certain cases in spite of good healing and take of the grafts. Analysis of these instances revealed that if the burned area extended beyond the dorsum of the hand and involved the wrist as well, finger flexion was always more limited. It does not appear likely that tendon involvement is responsible since wrist flexion was not limited proportionately. Normally flexion of the metacarpophalangeal joints results in distal movement of the skin over the wrist and hand as much as six to eight millimeters. Restriction of this skin movement which should start two to three centimeters proximal to the wrist is the probable mechanism of the prolonged finger disability.

The return of function already obtained in these patients under treatment for a relatively short period is encouraging. There appears to have been minimal permanent joint or tendon injury and the prognosis seems good for eventually securing excellent function of the hands.

## THE PROBLEM OF BURN SHOCK COMPLICATED BY PULMONARY DAMAGE\*

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The treatment of shock of the casualties demands priority in a disaster. Proper immediate shock therapy may prevent early death among burn casualties and also influence the future course because the fight against infection depends upon the patient's well being.

The pulmonary lesions in the casualties of the Cocoanut Grove disaster were unexpected and complicated the care of the shock. The usual plan of action for the care of shock due to burns had to be promptly modified. Carbon monoxide poisoning with its bright cherry red color of the burn surfaces and mucous membranes and the inflammation of the burned lungs and airways were quickly detected but there was a delay in recognizing that the resulting anoxia was the cause of the mania in some of the patients. Dr. Beecher in his article deals with the problems of anoxia. In this article we detail the modifications deemed necessary for the care of the burn shock.

A disaster close to hospital facilities offers the ideal circumstances for the care of shock. For the prevention of shock is more effective than its treatment when once it is established. This is particularly true of burn casualties where the shock, except for the primary phase due to pain and exposure, has a longer latent period than shock from hemorrhage. The Cocoanut Grove night club was sufficiently close to the Massachusetts General Hospital so that all the casualties arrived within two hours of the onset of the fire. The shock that had been suffered by the time of arrival was due not to burns so much as to the anoxia, exposure and pain. The steps taken in the treatment of shock were as follows:

*Control of Pain*—Each patient immediately upon admission was given an injection of morphine. This procedure routine in the treatment of burns, is based on the concept that prolonged pain in itself leads to shock. That a few of the patients received an overdosage from a mistaken idea of therapy is emphasized by Dr. Beecher.

That pain may exert an additional and indirect influence on shock is illustrated by Case 12, a young naval officer. One of the earliest patients to arrive, he walked in. There was a delay before he received the morphine. The pain in his hands was so intense that he was unable to lie down but jumped up and down on the floor waving his hands. Twenty five hours later he died of the pulmonary complication. It is a question whether this initial excessive physical exertion may not have increased the pulmonary edema. It is a common belief as a result of experiences in the last war that exertion precipitates the onset of pulmonary symptoms and edema following phosgene inhalation.

\* The work described in this paper was done under a contract, recommended by the Committee on Medical Research, between the Office of Scientific Research and Development and Harvard University.

General anesthesia was deliberately omitted as part of the treatment of the casualties since it has a deleterious influence in patients in impending shock. The surface treatment, with no débridement and no cleansing, was planned with this in mind.

*Minimum of Manipulation*—Every effort was made to reduce manipulation in the care of the patient. Rolling a patient over abruptly tends to disturb the vascular equilibrium with which he is responding to the diminishing blood volume. But one shift of the patient was made, from stretcher to bed. All subsequent procedures were carried out in bed. Had débridement and cleansing been performed it would have necessitated a transfer of patient from bed to operating table and back, as well as the manipulation incident to the débridement.

*Plasma Therapy*—All except ten of the patients were given plasma intravenously. Of those receiving no plasma, five had minor pulmonary complications and no surface burns, and were discharged from the hospital in the first few days (Cases 9, 10, 21, 30, and 31), the other five had minor surface burns and various degrees of pulmonary signs (Cases 1, 3, 5, 15, and 24). Because of the inevitable delay in the thawing of plasma in the blood bank, the early arrivals did not receive plasma promptly, whereas the later arrivals had plasma running into their veins within five minutes of entry. Plasma therapy was deliberately withheld from the patients with early signs of pulmonary damage and no, or minor, external burns. To one patient, (Case 6) with no external burn but severe pulmonary damage, one unit of plasma was given later in the night.

The initial dosage of plasma was determined on the basis of the surface area of the burns\*. For each 10 per cent of the body surface involved, it was planned to give 500 cc in the first 24 hours. Because the plasma delivered by the Blood Bank during the first 36 hours was diluted with an equal volume of physiologic saline solution, the patient was to receive 1000 cc of fluid for each 10 per cent burned.

The plasma dosage was modified subsequently on the basis of repeated hematocrit and serum protein determinations. The hematocrit readings on blood taken in the third hour after entry were available by 3 A M of the first morning. The rate of plasma administration was increased in the patients showing hemoconcentration. Four additional blood hematocrit and protein determinations were made in the first 24 hours. Three determinations were made in the second 24 hours, two in the third, and daily thereafter as indicated. No attempt was made to apply a formula to the hematocrit reading to determine the dosage of plasma needed, it was simply run in faster when hemoconcentration was present. The same basic formula of surface area

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\* No attempt was made to judge the amount of plasma which might be lost from the circulation into the damaged pulmonary tissue. That the lungs accounted for some of the plasma lost is indicated by the fact that those lungs examined postmortem by Drs Mallory and Brickley weighed nearly one kilogram more than an average pair of normal lungs.

was adhered to \* Only two patients (Cases 4 and 39) showed a hematocrit reading below normal in the first 24 hours

The results of this intravenous plasma saline therapy were apparently satisfactory in the initial 12 hours Although forms of shock certainly existed little or no burn shock was encountered† No patient died in the first 12 hours (The unknown number who died immediately at the entrance from suffocation are excluded) The seven deaths counted occurred from 13 to 62 hours after admission and all were considered to have been caused by pulmonary damage and anoxia

Little or no hemoconcentration as judged by the hematocrit and serum protein occurred in the patients with the most extensive surface burns For example in three of these patients (Cases 13, 20 and 34) the highest recorded hematocrits were 53, 55 and 54 per cent The hematocrit of a severely burned female (Case 8) did reach 65 per cent the highest recorded figure

Unsuspected hemoconcentration appeared in patients with pulmonary damage and lesser external burns The hematocrit of two of these (Cases 5 and 19) reached 55 and 59 per cent This discrepancy was undoubtedly due, in part to the fact that more attention was paid to prompt plasma administration to those who were severely burned It is evidence on the other hand of the early loss of plasma into the damaged pulmonary bed

All patients showing a severe degree of the typical burn pattern burns of the head and hands developed hemoconcentration and eventually required more plasma in the first 24 hour period than had been calculated by the surface area formula Massive edema formed beneath the deep burns of the face and scalp in spite of the pressure dressings The edema fluid was dispersed downward to the neck and over the shoulders and chest Apparently in the deeper burns there was a correspondingly deeper damage of the subcutaneous capillary bed with an increasing extravasation of plasma into the loose areolar tissues beneath face, scalp and downward into the fascial planes of the neck In the mild burns of the face, such massive edema did not occur and hemoconcentration was not excessive

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\* When the hematocrit reading was 60 per cent or over three units of plasma were given in the four hours before the next hematocrit reading If the hematocrit was 55 to 59 per cent, two units were injected, if 50 to 54 per cent, one unit

† Burn shock is defined as low blood pressure shock, with hemoconcentration and diminished blood volume due to loss of plasma fluid into the burn area Blood pressures below 100 mm Hg were recorded on several of the patients in the first 12 hours One man (Case 26) entered with a blood pressure of 80 mm He had been sprayed with water and was chilled His pressure recovered spontaneously within 30 minutes The woman (Case 2) who had severe anoxia in the first seven hours in part due to an overdose of morphine, and who received artificial respiration through an intratracheal tube, showed an irregular blood pressure curve with several systolic readings below 60 and one of 70 (She developed auricular fibrillation) The other patients with low blood pressures exhibited them for the most part, for only short period Dr Aub, seeking to investigate shock, was unable to find a patient with a sufficiently low pressure for a long enough time during the first night to make study worthwhile Blood pressures were recorded during this time in all except two patients (Cases 13 and 34)

In spite of the possible inadequacy of the surface area formula when it is applied to burns of the head and scalp, this formula has much to recommend it. It is true that different degrees of burn may result in different amounts of edema but it is still true, roughly, that the amount of plasma fluid lost from the circulation into the tissue spaces is proportional to the area of the burn. The formula is simple to calculate and can be done rapidly, facilitating the handling of many patients. No blood determinations are necessary and it can be applied when laboratory facilities are not available. The complicated formulae of Harkins,<sup>1</sup> and Elkinton, Wolff and Lee<sup>2</sup> are not satisfactory merely because laboratory determinations and calculations are necessary in order to apply them.

There is a fundamental difficulty to the use of these formulae and also to that of Black. These three formulae tell at the specific moment the blood determination is made how much plasma is required to bring the plasma volume back to normal within a short interval. They do not allow for the amount of plasma that will leak out of the circulation over the ensuing hours. If one of these formulae is applied soon after the burn, when the patient has just entered the hospital, and but little hemoconcentration has occurred, an inadequate amount of plasma will be given. On the other hand, if the formula is applied when the maximum edema has already occurred, an unnecessarily large amount of plasma may be injected, for at that time the rate of plasma loss is diminished. Any formula made on a given blood determination must also take into account the expected loss in the hours to come and this can only be estimated on the basis of the surface area burned. A combination of the two methods is the best.

A difficulty which was partly anticipated but not adequately solved in these patients was the problem of the amount of plasma and intravenous therapy required by a patient with pulmonary damage. After the arrival of the first 15 patients it was obvious that some type of pulmonary pathology was present. At first sight, it was thought that there must have been an explosion and that the pulmonary damage was due to the blast. The intravenous solutions of saline and glucose which were already running into the patients were slowed to a minimum while awaiting delivery of the plasma from the bank. This was done in an effort to prevent the appearance of pulmonary edema in the damaged lungs.\* Already several casualties,

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\* Both sodium chloride and glucose are freely diffusible through the capillary wall in contrast to the plasma proteins which are only partially or slowly diffusible. In the area of a burn or chemical inflammation, the capillary permeability is increased and plasma proteins pass out more freely into the extracellular spaces along with the water and electrolytes to form the edema fluid. Saline or glucose solutions, by raising blood pressures in burned patients, tend to wash more plasma protein out into the area of injury and increase the edema. Since only a portion of the plasma proteins leak out of the capillary, an injection of plasma is more efficient than saline in maintaining blood pressure in burn shock and if given slowly will not cause as rapid a formation of edema.

obviously suffering from anoxia had died within minutes after their arrival. As casualties arrived who were able to serve as reliable witnesses it became clear that there had been no explosion that only irritating fumes and heat were the cause of the pulmonary inflammation †

In spite of the early signs of pulmonary damage, plasma was given to most of the patients. Each patient was watched carefully and it was withheld from those who on the basis either of the mildness of their surface burns or lack of hemoconcentration apparently did not require it. With the progression of pulmonary signs however more caution was exercised. After three patients had died with signs of pulmonary edema the policy of allowing a certain amount of hemoconcentration to persist was resolutely adhered to in all patients with lung damage. The hematocrit readings were maintained around 50 per cent.

Whether the dehydration regimen was of benefit is questionable. It was not sufficiently severe to eliminate kidney function and perhaps was of no detriment. On the other hand four more patients died with signs similar to those of the earlier three. In three of these latter patients postmortem examination revealed damage incompatible with life due to tissue damage in the bronchioles rather than to edema. It is still an unsettled point but if we were faced with the same condition again we would probably allow a little hemoconcentration to persist throughout the period of progressive pulmonary signs.

Normal kidney function was maintained in all but three patients in spite of the hemoconcentration which was allowed to the patients with pulmonary complications. Normal kidney function is assumed when the blood non-protein nitrogen was within normal limits the urine volume adequate and the disappearance of hemoglobinuria and albuminuria at the end of two weeks. No renal function tests were performed.

The nonprotein nitrogen of the blood was determined daily from the second through the fifth day in all patients who remained in the hospital and then every other day or as indicated. Nonprotein nitrogen values above normal were recorded on six patients. In one patient (Case 38) who vomited nearly everything taken by mouth for the first six days abnormal nonprotein nitrogens of 51 and 62 mg. were recorded on the third and fourth days. In two of the patients who died on the third day (Cases 25 and 27) the nonprotein nitrogen rose just before death to 64 and 78 mg. These are the three

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From the same point of view the physiologic saline solution used in equal volume to dilute the plasma was omitted after the first 36 hours. Fifty per cent glucose was substituted, 50 cc. for each 150 cc. of plasma, in order to maintain the free flow of the plasma. (The flow of undiluted plasma is sluggish.)

† The similarity of the pulmonary signs encountered in this disaster to those of the Cleveland Clinic fire of 1929 was not at first apparent. We had thought of that catastrophe as unique and that with the change in chemical composition of roentgenographic films similar irritating poisonous nitrogen gases would not again be encountered in civilian life. The clinical course of these patients was also comparable to that seen in soldiers following phosgene inhalation in War I.

patients considered to have had diminished renal function. Two of these three patients had additional signs of abnormal kidney function. Case 38 showed albuminuria and Case 27 both albuminuria and massive hemoglobinuria. In Case 25 the urine findings were normal.

The other three patients in whom an elevated nonprotein nitrogen was recorded were Cases 4, 8, and 36. In Cases 4 and 36 the nonprotein nitrogens were elevated on a single occasion, 62 and 44 mg, respectively, on the fourth day. The hematocrit and serum protein determinations on

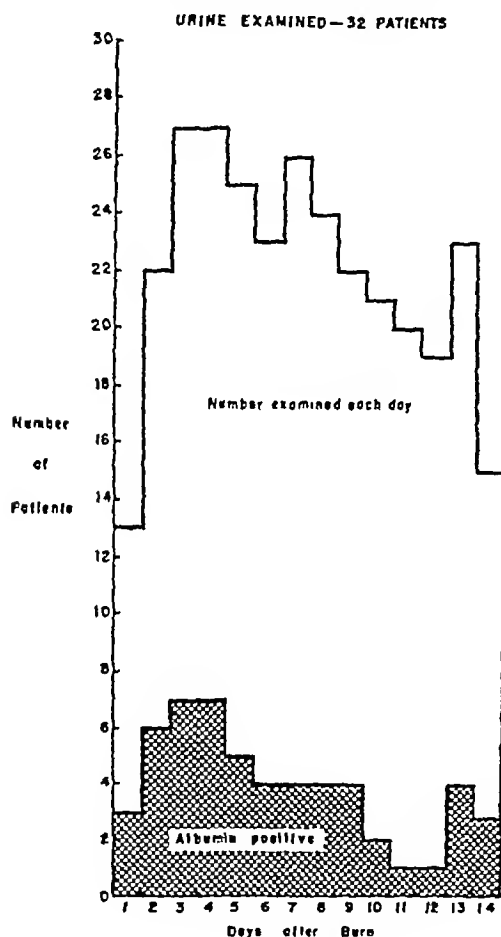


FIG. 43—The occurrence of albuminuria in 32 patients

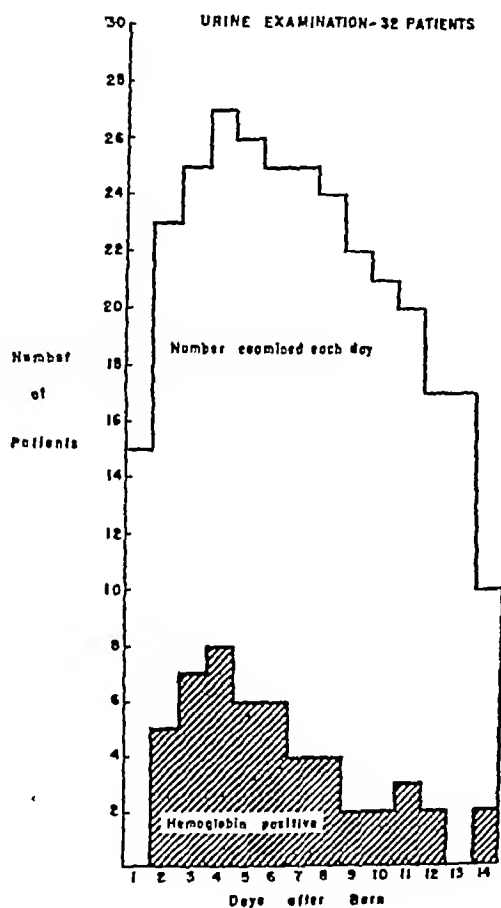


FIG. 44—The occurrence of hemoglobinuria in 32 patients

both of these patients were normal on that day. The final abnormal non-protein nitrogen reading was 51 mg in Case 8 on the seventeenth day, the hematocrit was 35 per cent and the serum protein 7.3 mg. The patient had received a transfusion the day before and received another on each of the next two days because of the low oxygen carrying capacity of the blood. In none of these three patients were there other signs of abnormal kidney function such as albuminuria, hemoglobinuria or diminished urine volume.

Albuminuria occurred in 12 patients, see Figure 43. This includes one of the patients who died. No patient showed albuminuria after the second week. It is noteworthy that the period of albuminuria coincides with the period of resorption of edema of the burns. It cannot be con-

cluded however that the protein of the edema fluid is necessarily excreted by the kidney as albumin for several of the patients with massive edema exhibited no albuminuria.\*

There was no absolute correlation between the albuminuria and the type of lesion. It appeared in six of the nine patients surviving with extensive burns and in one who did not. It was found in the girl with pulmonary damage and no external burns (Case 6) and in three patients with minor burns one of whom was jaundiced. It also occurred in a man with moderate burns and pulmonary damage.

Hemoglobinuria was recorded in nine patients. In one who showed a mild degree it was probably caused by the sulfadiazine for there were concomitant crystals and this patient alone of the nine did not show albuminuria. In the other eight no sulfadiazine crystals appeared during the period of hemoglobinuria. In these eight the time incidence was much the same as that of the albuminuria (Fig. 44). Five of the eight had massive hemoglobinuria the urine was grossly dark brown to almost black. One who had it (Case 27) died on the third day. The other three showed only slight to mild amounts of hemoglobinuria and it is possible that in these the sulfadiazine may have been the cause except that the hemoglobinuria ceased even though the sulfadiazine was continued. Since but one of the group died and none of the surviving patients has shown any clinical evidence of impairment of renal function hemoglobinuria *per se* does not necessarily result in renal damage.

It is probable that the hemoglobinuria in the patient who died was a coincidence rather than a contributing cause of death. At postmortem examination of this patient the kidneys were found to be congested with occasional petechial hemorrhages but there was no evidence of renal damage.

Experimental evidence is accumulating to show that the liver plays an important role in the body's compensation to shock. An examination of liver function therefore following a shocking procedure such as a burn may be of importance in determining the character of a patient's response. Measurement of the prothrombin time was made on all patients on the third and fourth day and subsequently in a few in only three patients was the time prolonged. In one man who was severely burned (Case 11) on the fourth day the clotting time was 30 seconds that of the control 20. These identical findings were recorded on Case 32 on the same day on the following day she was jaundiced with a van den Bergh of 7.3 mg. The

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\* The coincidence of albuminuria and resorption of edema is a strong point in favor of the protein of the edema fluid being the source of the albumin. Dr. Zamecnik, at this hospital has determined increased peptidase activity in human bleb fluid from burn blebs and in lymph flowing from the burns of dogs. Peptidases might alter the proteins of the edema fluid in such a way that they would be excreted by the kidney rather than reutilized. That the greater portion of the extravasated protein is made use of in the organism rather than excreted is proven by the decreasing excretion of nitrogen by the kidney during the period of resorption of edema. (See Nitrogen Balance under Metabolic Observations)



third patient with an abnormal prothrombin was Case 27, who on the third day, the day of death, had a time of 36 seconds, the control 24

The patient recorded above with the elevated van den Bergh was the sole patient exhibiting jaundice. She had had a severe and prolonged anoxia resulting in damage to the central nervous system. The initial attack of jaundice disappeared completely in a few days but a month later, when she was receiving no sulfadiazine, she had a recurrence of jaundice with a palpable enlargement of the liver. This attack also subsided but not until after two weeks. The origin of this jaundice and hepatitis was undetermined. It can be postulated that it was initiated by prolonged anoxia.

Hemolysis was observed in only one patient (Case 34). She was extensively and deeply burned with large areas of charred skin. She died on the second day. The origin of such hemolysis is not clear. It is possible that it is the result of the actual heating of the blood present in the tissues at the time of the burn.\*

Intestinal ulceration and bleeding are common sequelae of burn and other forms of shock. The origin of the lesions which give rise to the bleeding is not clear. It is possible that they are the result of anoxia of the mucosal surfaces due to capillary stasis. The stasis may be due to hemoconcentration with increased viscosity of the blood or to diminished blood flow following arteriolar constriction. Arteriolar constriction and diminished blood flow are known to occur in various parts of the body as compensation for the diminished circulating blood volume and are an effort on the part of the body to diminish the capacity of the vascular bed to make up for the shortage of available blood volume.

In either case the incidence of intestinal ulceration and hemorrhage might be considered an index of successful shock therapy. Figure 45 shows the number of stools with a positive guaiac test. None of the stools was grossly bloody or tarry. It is interesting that Case 38, with the prolonged vomiting, did not show blood. Those which were positive for blood were found in patients with all of the different types of lesions. The largest amount of blood recorded was in Case 20, who showed a +++ test on the seventh day. At this time the hematocrit was below normal and whole blood transfusions were subsequently required. She had extensive deep surface burns and it was not believed that the loss of blood in the intestinal tract significantly contributed to the progressive anemia.

An unexpected finding of interest was the occurrence of alkalosis in two patients. On both the third and fifth days, an arterial puncture was done on Case 6, the young girl with pulmonary lesion and no surface burns. The  $pH$ 's were 7.63 and 7.40, and on the first blood the  $CO_2$  was 30.3 m.eq.

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\* In the experimental laboratory we have learned to associate hemolysis with the severity of the burn.<sup>3</sup> Hemolysis is not encountered in dogs with hot water burns of the extremities when the temperature of the water is less than 100° C. or when at 100° C. it is applied for 15 seconds or less. At 100° C. it appears if the burn is for 20 seconds, and invariably if the burn is 30 seconds or longer.

and the oxygen content 17 vols per cent. On the second blood the sodium was depressed at 138.6 meq whereas the other findings were normal. Total base 152.5 meq calcium 9.0 mg chloride 100 meq phosphate 4.3 mg phosphate 4.3 mg and hemoglobin 12.7 Gm.

A second patient (Case 13) with both extensive severe skin burns and moderately severe pulmonary complications showed an alkalosis as well as an anemia on the fifth day. An arterial puncture was made to determine the oxygen content because of the falling hematocrit and progressive anemia.

#### STOOL EXAMINATION—25 PATIENTS

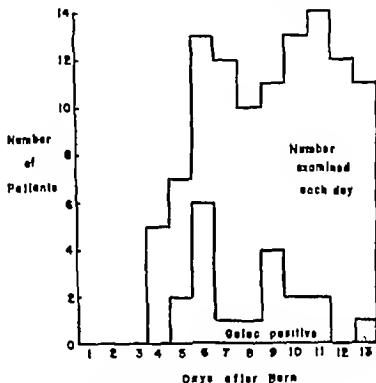


FIG. 45—The occurrence of blood in the stool of 25 patients.

The hematocrit was 38 per cent hemoglobin 9.4 Gm and  $pH$  7.47 non protein nitrogen 24 mg the total base 147 meq sodium 133 meq calcium 9.3 mg chloride 97 meq  $CO_2$  content 28.5 meq phosphate 1.7 mg. The phosphate was low presumably because the patient had eaten recently. The oxygen capacity was 14.6 vol per cent content 13.1 vol per cent or a saturation of 89.8 per cent.

The alkalosis in these patients was due presumably to the sulfadiazine therapy<sup>4</sup> and not to the pulmonary complication. For the probable origin of the progressive anemia see below under *Maintenance of Nutrition*.

**Chemotherapy**—Chemotherapy to effect bacteriostasis is an integral part of the modern treatment of burns. At the moment the sulfonamide preparations prevail unfortunately they occasionally lead to untoward results and disturb organ function and metabolism. Since the maintenance of normal bodily function is part of good shock therapy any discussion of the treat-

ment of shock in burns must needs include chemotherapy. It has been pointed out above that sulfonamide therapy leads to a loss of base and to alkalosis but this is apparently of little import. It is the kidney and liver damage which occasionally occur even without overdosage which are of greatest significance.

The relation of infection to shock in burn patients has been but little explored scientifically. Our present clinical impression is that infection is often culpable. It is possible that the shock, even as soon as in the late hours of the first day, is in part due to the toxemia of bacterial infection. Certainly in later days infection supersedes in responsibility all other factors. The malnutrition and anemia (see under *Maintenance of Nutrition*) are almost certainly of infectious origin.

There is also a reciprocity between shock and infection. In shock, due to a disproportion between the available circulating blood volume and capacity of the circulation resulting in decreased blood flow to tissues, anoxia inevitably develops. It is well known that certain organisms, including the *beta* hemolytic streptococci, multiply faster in even slight degrees of anoxia. In burn wounds, therefore, in which any degree of anoxia exists, organisms multiply more rapidly and infectious toxemia develops relatively early.

Such thoughts only emphasize the importance of minimizing bacterial contamination of the burn wounds by immediate coverage of the surface.\*

*Maintenance of Nutrition*—In the treatment of burn shock every effort should be made to administer the necessary fluid and protein through the gastro-intestinal tract. Patients with less than ten per cent of the body surface burned, particularly if mildly, and if they are not nauseated or vomiting, may be treated by this route. They should drink readily digestible fluids with a high protein content. Milk and milk products, high protein bouillons with amino-acids added are such fluids. Warning should again be made that if there are deep burns of the face and scalp, particularly if there are acute pulmonary lesions as well, there may be an unexpectedly great loss of plasma volume and intravenous plasma therapy may be required. Ten of the patients, some with mild burns, and some with pulmonary damage only, were treated entirely by mouth.

High protein therapy by mouth is not sufficient. A caloric intake sufficient for maintenance is advisable. It is probable that a moderate carbohydrate intake is required for normal liver function.

Attention to the necessary accessory food substances is also imperative. All of the patients received large doses of the vitamins both as natural and synthetic substances, from the first day. In some, these substances had to be given intravenously. It is to be remembered that patients with severe burns even with minimal infection, have fevers, that their metabolic rates

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\* The availability of sulfonamides to the burn wound has been considered in a previous paper on the surface treatment. Sulfonamide levels in bleb fluid were recorded on five patients (Cases 17, 19, 20, 28 and 38). Levels together with the simultaneous blood levels are available in the protocols.

are therefore elevated and their requirements both for total calories and accessory substances are increased. For the patients with more active infection this is the more true.

None of the patients developed clinical signs of a vitamin deficiency. They were not weighed on entry but of the 11 patients with severe burns who survived, almost all lost some weight and two lost a very considerable amount (Cases 11 and 13).

Constant attention was paid to the development of the initial signs of anemia. None of the patients with second degree burns developed clinically significant anemia and none received therefore a whole blood transfusion except the woman in whom anoxic damage to the central nervous system occurred. Eight of the ten patients with third degree burns who stayed at this hospital until healed received transfusions for anemia. In three one transfusion sufficed. Another three patients received three, four and five transfusions each. The most severely burned patient to survive (Case 13) received in all 25 whole blood transfusions of 500 cc. With this latter patient we faced difficulties.

Her burns were so extensive and the change of dressings so painful that endless time was consumed in clearing the areas of dead tissue. From the fifteenth to forty fifth days efforts were made to change all of the dressings at one time and general anesthesia was required each time the dressing was done. The patient was nauseated for each following 24 hours. The nutritional status of the patient suffered and her courage failed. Intravenous amino-acid injections as well as whole blood transfusions were given by femoral vein. (A single vein in one arm was the only other available vein in an extremity). It was not until the dressings were assigned to a single team to be done without general anesthesia for lesser areas at one time that the patient's nutritional status improved. With the advance in general health, epithelial proliferation across granulating areas became obvious and grafting was possible.

This patient presented psychotic tendencies and it was not until a rapport was reached between the patient and Dr. Cannon and his two intern assistants that mastery of the nutritional state was achieved. The rapport was abetted by the withdrawal of other physicians who previously had had jurisdiction over aspects of her care. The importance of the psychologic factor in the production of good digestion cannot be too strongly emphasized. All too frequently where there are many doctors, each responsible for a different aspect of the patient's regimen the patient is unable to find one physician to whom she can turn.

*Adrenal Cortical Extract*—Extract of the adrenal cortex containing the active principle of the gland has been recommended in the treatment of burn shock<sup>6, 7</sup>. The predominant signs and symptoms of adrenal cortical insufficiency both in man and animal are those of shock. Among other findings there are a low blood pressure, hemoconcentration and an elevation of potassium and phosphate in the blood. Such findings are also characteristic of shock appearing after trauma, burns, intestinal obstruction and sometimes severe infection.

The problem of the relation of the adrenal cortex to burn shock has been under investigation in the Surgical Research Laboratory during the past year.

# COPE AND RHINELANDER

It has been found that in adrenal insufficiency in the dog there is a generalized increase in capillary permeability. This increased permeability is reversible by the administration of adrenal cortical extract.<sup>7</sup> In the dog there is also an increase in capillary permeability in the experimental burned area as has been demonstrated by Field, Drinker and White,<sup>8</sup> (1931), and more recently by Glenn, Peterson and Drinker<sup>9</sup> (1942). In our laboratory this increase in permeability has been shown to be localized to the region of the burn.<sup>10</sup> Only rarely does an increase in the nonburned area develop following prolonged shock. Efforts to decrease this abnormal permeability by large doses of adrenal cortical extract have failed. Although under different experimental conditions it might be possible to influence the abnormal permeability induced by a mild burn, certainly the adrenal cortical hormone, judged by these experimental results, would appear to have little practical usefulness in burn patients.

From time to time, in this hospital, selected patients with burns have been treated with adrenal cortical hormone in the effort to evaluate this substance. No unequivocal benefit has been obtained from its use. Two of the patients of the Coconut Grove were selected for adrenal cortical therapy (Cases 34 and 27). Both of these patients had extensive surface burns and pulmonary injury.

On the afternoon of the first day, when it was clear that Case 34 had severe pulmonary damage, adrenal cortical extract (Upjohn) was started, 50 cc were given intravenously over a one-hour period. The patient's blood findings before and after receiving the extract are given in Figure 46. She had already received eight units of plasma, and only a minor amount of hemoconcentration had occurred. She was selected for extract therapy in the hope of decreasing pulmonary edema by reducing the capillary permeability in the pulmonary bed.

The patient died six hours after receiving the extract. The course had been progressively downhill, with increasing anoxia in spite of oxygen administration. There was no clinical evidence that the patient had benefited from the extract. Admittedly, the patient's disease was profound and this was a rigorous test for any mode of therapy.

The second patient (Case 27) was also started on extract therapy on the first day, 14 hours after the fire. The blood determinations, before and after

CASE 34

Date	Time	Hemat %	Pl Prot Gm %	Na m Eq/l	Cl m Eq/l	Therapy
11/29/42	2 A.M.	51				
	5	50	6.8	139	103	Plasma 3 units
	10	52	7.6			Plasma 3 units
	4 P.M.	51	7.0			Plasma 2 units
	5 25					ACI started
	6 25					ACF 50 cc finished
	10	54	7.0	102		
11/30/42	12 50 A.M.		DIED			

FIG. 46.—Adrenal Cortical Extract (ACI) Schedule of administration together with plasma injections and blood findings in Case 34.

## SHOCK

extract are given in Figure 47. Before and during the extract therapy the patient received eight units of plasma. Over a period of 14 hours 70 cc of extract were given. She died 35 hours after the last injection of extract. During that period she received an additional five units of plasma. The nonprotein nitrogen which was 28 mg at the time of administration of the extract rose to 90 mg before death.

This was another severe test for adrenal cortical extract. The patient had extensive deep surface burns and pulmonary damage; anoxia persisted in spite of an oxygen tent. This patient too was chosen to treat with extract because of the pulmonary lesion in the hope that the extract would diminish capillary permeability in the lung bed and thereby reduce the edema. There was no evidence either during or subsequent to the administration of the extract that the edema was less or the respiration of the blood better.

The difficulty of clinically evaluating such a substance as adrenal cortical hormone in the therapy of burns or other forms of shock is apparent from the experience with these two patients of the Coconut Grove disaster. There were other patients with apparently as severe pulmonary injury or as extensive surface burns who survived without the administration of adrenal cortical extract. Had the two patients to whom the extract was administered survived it would have been as wrong to ascribe their survival to the adrenal hormone as it would be to incriminate the hormone as the cause of their death. There is no clinical evidence that the hormone in any way influenced the course of the disease.

### CASE 27

Date	Time	Hemat. %	PL Prot. Gm %	N.P.N. mg %	Cl mEq/l	K mEq/l	Dextrose mg %	Therapy
11/29/42	2:15 A.M.	56						Plasma 1 unit
	10	56	8.1					Plasma 1 unit
	12:40 P.M.							ACE started
	2:45							Plasma 2 units
	3:40							ACE 30 cc finished
	4	47	6.4			5.1		Plasma 2 unit
11/30/42	10	45	6.2					Plasma 2 units
	3:15 A.M.							ACE started
	2:45							ACE 20 cc. finished
	6	43	5.8					
	2 P.M.	44	5.9	28	102.7		8.2	
	8:45	42	5.3					Plasma 2 units
12/1/42	7:1 A.M.	35	5.6	78	113.9		7.0	Plasma 2 units
	12:15 P.M.							Plasma 2 units
	1:55							Plasma 1 unit

DIED

FIG. 47.—Adrenal Cortical Extract (ACE) Schedule of administration together with plasma injected and blood findings in Case 27.

### CONCLUSIONS

The pulmonary lesion in the casualties of the Coconut Grove disaster was unexpected and complicated the care of shock. Since such lesions may occur in civilian as well as military disasters, it is well to be prepared to meet them. The treatment of burn shock usually a straightforward problem of maintaining an adequate plasma volume had to be modified in an

effort to prevent excessive pulmonary edema. Anoxia, not ordinary surgical shock was the primary concern.

It was fortunate that a treatment of the burn surface had been chosen in advance which permitted a maximum of attention by the personnel available to the problem of shock. Plasma therapy was prompt and effective. There were no deaths in the first 12 hours. The seven deaths that occurred took place within 13 to 62 hours and were the result of the pulmonary complication.

An effort was made to diminish the edema in the damaged lungs by allowing a mild amount of hemoconcentration to persist.

Massive edema, out of proportion to the surface area involved, may occur beneath deep burns of the face and scalp. The use of the various formulae in computing plasma dosage is discussed.

Hemoglobinuria occurred in eight patients, one died with no anatomic evidence of renal damage. The other seven have survived with normal kidney function.

Adrenal cortical extract did not affect the pulmonary edema or the general bodily function of the two severely injured patients to whom it was administered.

#### REFERENCES

- <sup>1</sup> Harkins, H. N., Lam, C. R., and Romence, H. Plasma Therapy in Severe Burns. *Surg., Gynec. & Obst.*, 75, 410, 1942.
- <sup>2</sup> Elkinton, J. R., Wolff, W. A., and Lee, W. E. Plasma Transfusion in the Treatment of the Fluid Shift in Severe Burns. *ANNALS OF SURGERY*, 112, 150, 1940.
- <sup>3</sup> Cope, O., Mixer, G., Jr., and Graham, J. B. To be published.
- <sup>4</sup> Beckman, W. W., Rossemcisl, E. C., Pettengill, R. B., and Bauer, W. A Study of the Effects of Sulfanilamide on Acid-Base Metabolism. *J. Clin. Invest.*, 19, 635, 1940.
- <sup>5</sup> Rhoads, J. E., Wolff, W. A., and Lee, W. E. The Use of Adrenal Cortical Extract in the Treatment of Traumatic Shock of Burns. *ANNALS OF SURGERY*, 113, 955, 1941.
- <sup>6</sup> Scudder, J., and Elliott, R. H. E., Jr. Controlled Fluid Therapy in Burns. *South. Med. & Surg.*, 104, 651, 1942.
- <sup>7</sup> Cope, O., Moore, F. D., Mixer, G., Jr., and Graham, J. B. To be published.
- <sup>8</sup> Field, M. E., Drinker, C. K., and White, J. C. Lymph Pressures in Sterile Inflammation. *J. Exper. Med.*, 56, 363, 1932.
- <sup>9</sup> Glenn, W. W. L., Peterson, D. K., and Drinker, C. K. The Flow of Lymph from Burned Tissue. With Particular Reference to the Effects of Fibrin Formation upon Lymph Drainage and Composition. *Surgery*, 12, 685, 1942.
- <sup>10</sup> Cope, O., Moore, F. D., and Tobin, L. H. To be published.

#### A NOTE ON THE BLOOD BANK

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A BLOOD BANK is a requisite for the efficient handling of patients in a disaster. In the first place it is economical. In the second place, with burn casualties particularly, it is imperative to feel free to administer all the plasma necessary. There need be no restraint in the use of frozen plasma. Dried

plasma with its content of mercurial preservative offers a drawback since theoretically kidney damage may result if too much is given

*Supplies on Hand*—At the time of the Coconut Grove fire the Massachusetts General Hospital had 391 units of sterile frozen plasma in its own bank 38 flasks of whole blood 106 units of dried plasma and a reserve of 76 units of frozen plasma stored for emergency use at the Faulkner Hospital seven miles away This meant a total of 611 potential transfusions The frozen units stored in 500 cc Tenwal containers were diluted half and half with normal saline

For thawing we had three large tin pans in which the bottles could stand while a current of water at body temperature surged around them from a faucet. The temperature was controlled by hand with a water bath thermometer

In our central supply room were kept stored in dust proof containers 48 sterile packages consisting of intravenous tubing all connected to Tenwal vent tubes Baxter filters and glass adaptors In the same package were the usual shut off clamps two No 19 and No 18 needles in a test tube and an adaptor for making a connection with a needle to a Strumia bottle In our emergency supply room were a large number of poles to support infusion bottles as well as gauze splints and bandages

*System of Administration*—The administration of fluids began with a saline infusion started through an intravenous apparatus and later through the plasma sets with Baxter filters As soon as plasma was available 60 minutes after the arrival of the first victim these saline flasks were changed to plasma In many instances the plasma transfusions were started before the patients left the emergency ward to go upstairs to the floor cleared for their use A selected group of three interns was assigned to start transfusions and keep them going They went from bed to bed setting up apparatus or assisting the team in charge of the patient if it was having difficulty

*Veins Used*—The system of fluid administration at the hospital has in recent years been one of using the most peripheral veins first usually those on the back of the hands A sharp No 19 or No 20 needle is inserted for administration of saline and glucose, but at least a No 19 for transfusions Cannulae involving frequent subsequent destruction of veins are rarely used The extremity is carefully splinted and the needle well strapped to it. The needle is inserted with the aid of a glass adaptor rather than a syringe.

On the night of the fire this policy was employed but because of the number of arm and hand burns a very high percentage of foot and leg veins were used These are in an area difficult to splint They are often at an angle which impedes the insertion of the needle and subsequent fixation Furthermore, the situation was made worse by the patient being transported from the emergency ward by their restlessness and their being turned for the application of dressings It was necessary to cut down on the veins of eight patients during the first night and insert cannulae. They were only of temporary help particularly in instances where the plasma had already



run in, a decision had to be made whether more plasma was to be given, or saline or dextrose solution to keep the needle open. This also pertained to people with difficult veins who had a needle well inserted. It meant that some people received more saline than was wise.

*System of Filtering*—The filters occasioned us little or no trouble. Although we have found that whole blood undiluted by saline will stop in over five per cent of transfusions through Baxter filters, the dilute plasma used for these victims flowed well. In several instances, when the complaints of malfunctioning of the apparatus were laid at the door of the filter and it was changed and inspected, the amount of clot within it filled less than one-half of the filter and a change of the filter drip brought no improvement. In one case a patient received eight units through the same filter without stoppage.

Slow flowing after an initial rapid drip was noted in many instances. This was usually in leg veins which were small and tortuous. We blamed this slow flowing, when dextrose ran well, upon the relative gummy nature of the plasma. In other patients constricting pressure bandages or theoretically elevated venous pressure due to extensive pulmonary burns were blamed for this phenomenon. We inserted a three-way stop-cock in the line between filter and needle for Case 27, enabling us to use a syringe as a pump for more rapid infusion. This was successful, though it may tend to increase clot formation, and for whole blood would be dangerous because of cell fragility. We tried this in other instances and have now adopted it as routine for our plasma sets.

*Handling of the Supply*—The supply of thawed plasma was controlled by telephone communication with the bank. As the original 16 units were melted, more plasma was started and was sent to the wards as rapidly as possible. We tried to keep about ten flasks on hand at all times in the first six hours. After that the administration was not on an emergency basis, so that we only thawed what was needed. A total of 147 plasma transfusions was given: 120 in the first 24 hours, 19 in the second, and eight in the third.

The work in the blood bank was started by an able medical student on call for night groupings and cross-matchings. He was soon assisted by a blood bank nurse, two other nurses, and several interns. The melting pans are important. We now have two smaller ones, divided into 16 sections to keep the bottles from tipping, with an overflow to run into a sink. The records of the pools used were well kept in the bank, but, unfortunately, time was too pressing on the wards to note the number of each bottle administered to each patient.

*Remaking the Plasma*—There was an immediate appeal made for donors. This was greatly aided not only by radio announcements, but by the zealous action of two conscientious objectors, residents in the hospital for metabolic studies, who went out into a neighboring street, stopped passing traffic and asked for all to come in and give their blood. We had over 100 donors, many of whom had unfortunately been eating or drinking too recently, but

we had sterile sets enough to take 23 bloods. For the next week we received nothing but voluntary donors so as to remake our plasma. Because of a wonderful response and the help of the Red Cross which in the following week received more blood than they could ship and hence supplied many hospitals with it by the end of two weeks of very hard work we had made a total of nearly 300 flasks for our reserve for civilian defense. Ordinarily we make but 65 units a month. Our fears that use of plasma in a disaster would leave us depleted were unjustified.

*Follow-up Thoughts*—Our follow up on the work done gave the following results. First there were no reactions. That is there were no chills reported but possible temperature changes were masked by the pyrexia following the burns. There was but one patient with jaundice though others received plasma from the same pool without untoward effect. Second we felt that too much saline was given although the maximum administration to any patient was 2,250 cc. in 24 hours. It was possible that much more might have been given and that this amount of sodium chloride was inadvisable. We began to dilute our plasma with 50 cc. of 50% dextrose in the first pools made immediately following the disaster. Using fresh centrifuged blood these pools were being used within 38 hours after the fire. Third our 48 plasma sets complete with filters were too few for adequate handling of a large number of casualties. If we had not had such excellent service in obtaining resterilization we would have run short. Now the number has been doubled and stop-cocks inserted between filter and needle. Fourth the amount of fibrin clot was very small and was a nuisance in only two cases. Fifth we had some vein complications. The early ones were local infection as a result of improper care of the wounds in two cases and a slough for the same reason in the one sternal administration of plasma. The vein thromboses are discussed elsewhere.

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## A NOTE ON THE THROMBOPHILICITIS ENCOUNTERED

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VIRUALLY all of the factors which predispose to the formation of thrombi in the veins of the leg are present in a patient recovering from a burn. Shock with decreased blood flow and lowered venous pressure hemoconcentration with increased blood viscosity prolonged immobilization circular dressings injections into the veins of the leg sepsis edema and nutritional deficiency may all be expected to contribute to some degree. Of the 39 patients cared for in this hospital following the Coconut Grove disaster five presented thrombo-embolic phenomena of clinical significance. This represents an incidence of 13 per cent. However of the nine patients hospitalized for a prolonged period and requiring grafts to third degree burns,

four developed evidence of thrombosis of the leg veins, an incidence of about 45 per cent. Two of these presented roentgenologic evidences of a pulmonary infarct.

A word of explanation is necessary in regard to both the terminology used and the policy of treatment. The term "thrombophlebitis" is used to indicate an acute inflammatory process in the veins of the leg attended by local pain, tenderness, warmth and some systemic reaction. The term "phlebothrombosis" is used to cover those cases in which a bland clot is present in the veins, with minimal inflammation and adherence. In both groups the presence or absence of swelling depends on the level and extent of the involvement, gastrocnemius spasm ("Homan's sign") may or may not be present depending probably on the degree of perivascular reaction.

It has been the policy at this hospital for several years to treat both of these conditions by ligation and division of the involved veins, usually the superficial or common femoral vein. This ligation is carried out below the profunda femoris junction providing the common femoral is not involved. Where the thrombophlebitis involves the superficial saphenous system, ligation of the great saphenous at the saphenofemoral junction is considered adequate unless there is evidence of involvement of the deep system. Diagnostic roentgenograms, using the intravenous injection of diodrast ("venograms"), have not been made because experience has shown that these are often misleading and the procedure is irritating to the intima of the veins. Anticoagulants, such as heparin or dicoumarin, have not been used because it is felt that these do not affect either the established thrombus or infarct, that they only prevent further extension of thrombosis, and that, therefore, they do not guard against fatal embolism.

The patients of this series who showed thrombo-embolic phenomena are of considerable interest because they represent the whole spectrum of this group of diseases, ranging from the patient with pulmonary infarct and no local signs in the legs, to the patient with fulminating local thrombophlebitis but no pulmonary manifestations. Furthermore, they show the *modus operandi* of some of the predisposing factors in this group of conditions, factors which may be eliminated by more meticulous attention to details of the patients' regimen. There follows a brief narrative of the development of the venous complications in these five cases.

#### CASE REPORTS

Case 2—This was a slightly built woman, with poor muscular tone, who moved about in bed seldom during her convalescence unless urged to do so. The dressings on her right leg, where there were small second and third degree burns at the knee and ankle, were covered during most of her convalescence by a continuous spiral elastic bandage from toes to groin. However, her left leg was burned at the knee and despite precautions to the contrary she was found to have been dressed with a circular bandage with no lower-leg component on several occasions. At the time of entry, due to the presence of burns on both arms plus the fact that she was in an oxygen tent for some time, she had an intravenous infusion placed in her saphenous vein in the left ankle. Operative procedures to graft her left hand were carried out on December 26, January 9 and January 15. These included a skin graft which was taken from the left thigh; this donor site was dressed on one occasion with a tight circular bandage, later replaced with a bandage extending to the foot. At no time during this period were the patient's feet elevated, or was she on regular bicycle exercises.

On January 14 the Roentgenologic Department suggested that her previous pulmonary picture now showed a superimposed process most likely due to a small infarct, and on January 18 this was confirmed by the finding of a friction rub at the right base. Examination of the legs at this time was completely negative except for the superficial burns and wounds previously mentioned. However with established infarction in her right lung and no other demonstrable source for the emboli, it was felt that she had a phlebothrombosis in the calf veins of her legs and that ligation should be carried out.

Therefore, on January 19 a bilateral ligation of the femoral veins below the level of the profunda, was performed. At this level no clot was found and free bleeding was encountered from both directions. Following this operation no further infarcts were found clinically or roentgenologically and within a week her chest became flat and remained so until discharge four weeks later. When seen two months later her ligation wounds were well healed and she had no swelling or discomfort of any kind in her legs.

The phlebothrombosis presumably present in this patient resulted from many factors among which her prolonged immobilization and circular thigh bandage were outstanding. Although she was in shock for a period after entry and had received an injection in her saphenous vein the long (eight weeks) interval between these events and her infarcts suggests that they were not causes of the thrombotic process. Bicycle exercises or elevation of the foot of her bed as well as better bandaging might have helped ward off this complication. Once discovered the therapy seemed to be effective in preventing recurrence and her chest quickly cleared.

**Case 36.**—A man of 28, who was an active newspaper reporter and feature-writer prior to the fire was a vigorous and muscular male who in his early stay in bed was very active and moved about considerably. However as weeks passed he became progressively disinclined to move about. Burns on his hands were grafted on December 22 and January 2 and because of a desire to get him mobilized as soon as possible he was allowed to get out of bed and start walking again on January 7 five weeks after the fire. Despite repeated admonitions not to do so he persisted in sitting in a chair instead of walking about. This position, without footstool or leg elevation of any sort, resulted in dependency of his legs and possible pressure in the popliteal space. The dressings of his thigh donor sites on the left side were properly applied and carried down to the toes. One week after he had been allowed out of bed he complained of pain in his left leg. Examination showed tenderness in the calf and during the three hours after onset the size of this calf increased five-eighths of an inch. On the following morning these signs persisted and he had developed, in addition, an interesting variant of the Homans sign. Not only was the calf painful and limited in dorsiflexion, but also his gastrocnemius muscle was in such severe spasm that he had fully developed ankle clonus. The temperature of this leg was increased and his white blood cell count was 18,000. His temperature was normal. His lungs were negative on clinical examination.

A diagnosis of left thrombophlebitis was made and, on January 14, a left superficial femoral vein ligation was carried out, below the level of the profunda. The vein is opened routinely in this procedure and in this instance a large clot was removed by aspiration and free bleeding obtained. The clot had progressed upwards to within 1 cm. of the junction with the profunda femoris. There was considerable perivascular edema and thickening. His postoperative course was satisfactory and he was discharged 11 days later. At two months he had untoward leg symptoms.

The diagnosis of left-sided thrombophlebitis in this case was fully substantiated by the operative findings. The clot found was nonadherent at the level exposed, though presumably it was adherent lower down. According to our usual procedure the other leg should have had the same operation, but the signs were so clearly unilateral, that it was felt justifiable to limit the operation to the one side. Of the various predisposing factors in this case, the relation to his mobilization and habit of sitting in his chair, leaning forward and listening to the radio, stands out as most significant. The thigh bandage over the donor site and the long period in bed no doubt also played a part. He was never in shock nor did he have any intravenous infusions in his legs. His lack of pulmonary infarcts is probably traceable to the inflammatory nature of the process, with resultant adherence. The fact that the clot was free at the level of ligation, however, suggests that operation may well have prevented a subsequent massive embolus.

**Case 11**—A 24-year-old male, sustained extensive burns of his back. Because of pain he lay quietly most of the time. To facilitate moving about he was given a foot-board to press against, and a Balkan frame with hanging handles to lift himself, using his one good hand. On December 25, four weeks after the fire, he complained of chest pain and raised a little bloody sputum. However, his chest films were repeatedly negative and it was thought that his tracheal burn was giving rise to the blood-streaking. His legs were normal to examination save for the burn on the right knee. On January 5, grafts were taken from the left thigh and put on his arm and right knee. A snug circular bandage with no lower leg extension was applied to the knee graft. The next day this was discovered and rectified but in the interim the patient had had 18 hours of partial venous obstruction to his right leg. Within three days he developed soreness and swelling in the right calf. An exploration of his right femoral vein was made on January 8.

Because of the presence of unilateral signs and a unilateral causative mechanism (the tight dressing) it was considered permissible to depart from established precedent as in the previous case, and operate on only the one side. At operation the vein was found to be thickened and edematous, but with no clot at this level. Slow bleeding from below suggested a clot further down but none could be obtained with the Trendelenburg sucker.

He remained quite immobile in bed despite efforts to the contrary. On February 3 (nine weeks after the burn), he complained of pain in his left groin and rapidly developed a fever, palpable tender lymph nodes and a tender cord along the course of one of the thigh tributaries to the deep system. Despite these signs of an acute inflammatory process in the thigh he had no ankle edema or Homan's sign. The diagnosis of left-sided femoral thrombophlebitis was made and a left femoral exploration carried out.

At this operation an unusual situation was found. His left femoral vein was almost an inch in diameter, surrounded by considerable edema, with a wall 3 mm thick. Many large nodes, up to 2 cm in diameter, were in the femoral triangle. Large clots were aspirated from the superficial femoral and profunda branches. The common femoral vein was ligated above the entrance of the profunda. The saphenous was also tied because of involvement at its orifice, but tied proximal to the entrance of the superficial epigastric vein so that a continuous superficial channel was left running up to the epigastric system. Postoperatively the patient did well, and when, six to two months later he had no swelling or leg symptoms despite the extensive ligation of the left. He never developed any roentgenologic evidence of pulmonary emboli.

This patient evidently had phlebothrombosis of his right leg and later developed an acute thrombophlebitis in the left thigh. The right leg pathology was unquestionably furthered if not caused by the tightly applied dressing at the knee. The cause of the acute process on the left is hard to assign to any one factor but was undoubtedly a result chiefly of prolonged immobilization. The donor sites on the left leg were well healed at the time of onset of the acute thrombophlebitis.

**Case 8**—A 70-year old housewife was slightly built and of little natural muscular vigor. Left to her own resources she lay quietly in bed making no effort to move about. During her first 18 hours in the hospital she had had a cannula tied into her left long saphenous vein. She often had the head of her bed and the knee-rest raised with her legs slightly flexed. This produced pressure in the popliteal space and the flexion at the hips presumably increased the venous pressure in the legs. On December 26, four weeks after the fire she complained of pain in her left shoulder but her chest was bandaged and clinical examination was unsatisfactory. Therefore, a roentgenogram was taken, which was interpreted as consistent with an infarct at the left base, with possibly a second one behind the heart. Examination of her legs at this time was completely negative. Because of the fact that no other source for the emboli could be demonstrated it was felt that the presumptive source was the great veins of the leg. Therefore bilateral femoral vein ligation was decided upon.

This was carried out the same day and on opening the veins, no clots were found. Postoperatively she did well and developed no more infarcts. Two days after the roentgenogram mentioned above another one was taken which showed that she had developed a small amount of fluid at the left base making even more likely the previous diagnosis of infarct.

This patient falls into the group in which pulmonary embolism pointed to a thrombotic process in the legs and the absence of local signs made a diagnosis of phlebothrombosis presumptive. Immobilization and position in bed probably contributed as much to her troubles as any other factor. Although she had had a cannula tied into a saphenous vein an interval of four weeks had passed between this and the emboli and she never showed any saphenous pathology clinically. She had no burns on her legs and prior to her embolism had no grafts taken from the thighs.

**Case 14**—This patient developed a typical acute saphenous thrombophlebitis in his left leg eight days after the fire. He was lightly burned and had not been systemically ill since 48 hours after the fire and had been up and about the ward. However he had had an intravenous needle placed in his long saphenous vein at the ankle in his first few hours in the hospital. Through this vein had been infused about 1,000 cc. of 5 per cent glucose in saline, and 1,000 cc. of plasma. On December 7 he noticed a sore red streak up the leg from the intravenous site at the ankle. There was a palpable tender cord along the course of the saphenous vein. Therefore the saphenous vein was tied off at the saphenofemoral junction to guard against extension of this process up into the femoral and iliac system. The patient did well postoperatively his local process quieted down on rest and heat and three days later he was discharged to an Army hospital.

The diagnosis of left-sided thrombophlebitis in this case was fully substantiated by the operative findings. The clot found was nonadherent at the level exposed, though presumably it was adherent lower down. According to our usual procedure the other leg should have had the same operation, but the signs were so clearly unilateral, that it was felt justifiable to limit the operation to the one side. Of the various predisposing factors in this case, the relation to his mobilization and habit of sitting in his chair, leaning forward and listening to the radio, stands out as most significant. The thigh bandage over the donor site and the long period in bed no doubt also played a part. He was never in shock nor did he have any intravenous infusions in his legs. His lack of pulmonary infarcts is probably traceable to the inflammatory nature of the process, with resultant adherence. The fact that the clot was free at the level of ligation, however, suggests that operation may well have prevented a subsequent massive embolus.

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This patient is of interest in that he was the only one of many who received injections into ankle veins, who developed saphenous phlebitis

#### DISCUSSION

From time to time it became necessary to perform femoral vein punctures either to get blood for study, or to give intravenous infusions in patients in whom other veins were not available. This was done in seven patients (Cases 13, 40, 32, 27, 34, 23, and 8), in only one did thrombosis develop (Case 8), and there was such a long interval between the femoral vein puncture and the development of the thrombosis, that it is probable that there was no connection between the two. One of the patients (Case 13) is of especial interest because she had at least eight femoral vein punctures as well as extensive septic burns on both legs. She never developed any evidence of pulmonary infarction. Examination of the calf veins was impossible due to the overlying burns.

The presence of burns or sepsis in the legs does not seem to predispose to thrombosis providing, of course, that the sepsis does not progress to the point of involvement of the larger blood vessels. However, lesions on the legs may be an indirect cause of venous complications if tight circular bandages are used which obstruct and distend the venous system distal to them. Of the five patients in this group with thromboses, only two had leg burns.

#### CONCLUSIONS

It is felt that patients who have been severely burned are prone, in a rather high percentage of cases, to develop thrombo-embolic phenomena from the veins of the legs. Constant vigilance must be exercised particularly in relation to prevention. The position of the patient in bed, and mobilization in bed with measures taken to ensure adequate venous circulation, are important factors in preventing thrombosis and embolism. When leg signs are positive, or evidence of pulmonary infarction is present, the treatment of choice is immediate ligation of the involved veins.

## METABOLIC OBSERVATIONS\*

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A study of the nitrogen metabolism and its relation to the adrenal cortex and 17 ketosteroids in burn shock was already under way in these laboratories prior to the Coconut Grove disaster in the hope of elucidating some of the problems under discussion in recent years regarding the body's compensation to injury. Selye<sup>1</sup> working with experimental animals has called such compensation the alarm reaction and points out that part of this reaction is an enlargement of the cortex of the adrenal gland.

An aspect of the compensation to injury encountered both in the human being and experimental animals is a sustained rise in blood sugar in spite of fasting. There is apparently an increased production of glucose from noncarbohydrate sources as well as from glycogen. In human beings following a burn such a rise in blood sugar occurs. There is also an increased excretion of nitrogen and 17 ketosteroids in the urine and it has been postulated that these three findings are signs of augmented activity of the adrenal cortex part of an 'alarm reaction'.

Several laboratories have observed an increased renal excretion of nitrogen in the human being following a burn.<sup>2,3,4</sup> The amount of nitrogen may be considerable, indeed comparable to that encountered in diabetic acidosis. In this latter disease it is believed that there is an overproduction of glucose and ketone bodies from protein; this excessive conversion of protein is perhaps motivated by the anterior pituitary uninhibited by an adequate supply of insulin. In the burned patient it has been postulated that the excessive nitrogen excretion is the result of increased conversion of protein to sugar but motivated in this case by the adrenal cortex.<sup>3,5</sup>

Long<sup>6</sup> has produced evidence in experimental animals that the adrenal

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\* The work described in this paper was done under a contract, recommended by the Committee on Medical Research, between the Office of Scientific Research and Development and Harvard University.

† The term "17 ketosteroid" is applied to those organic compounds which have a ketonic radical attached to the 17-carbon atom of a phenanthrenecyclopentane nucleus. Although this nucleus is common to such sterols as cholesterol, the sex hormones, the adrenal cortical hormones so far identified and members of the vitamin D group, the 17 ketosteroids which are excreted in the urine appear to represent, for the most part, metabolic end products of the androgenic hormones. These end products originate both in the testis and the adrenal cortex of both sexes. They do not represent, apparently, the entire biologically active secretion of the adrenal cortex. The non-17-ketosteroid excretion product of the adrenal cortical secretion, the so-called "cortin-like" sterols, can be measured only by a difficult biologic assay. The 17 ketosteroids, on the other hand, are determined in the urine by a quantitative colorimetric method and, even though in the male it registers the function of two glands, it is clinically the most useful method available by which adrenal cortical function may be estimated.

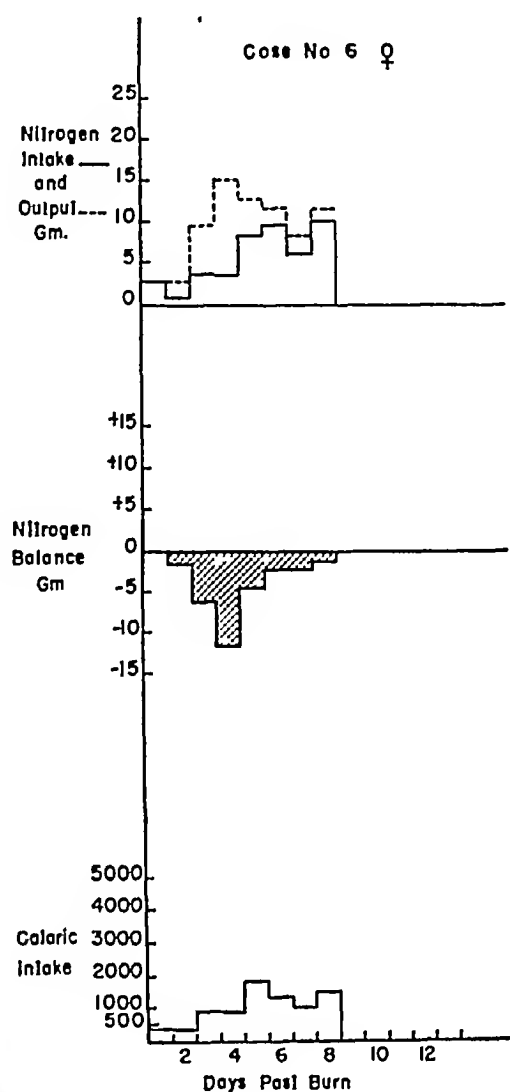


FIG 48—Nitrogen balance and caloric intake of Case 6, a girl with pulmonary damage and no surface burns. She required oxygen therapy for five days. She was afebrile throughout. One unit of plasma was given on the first day.

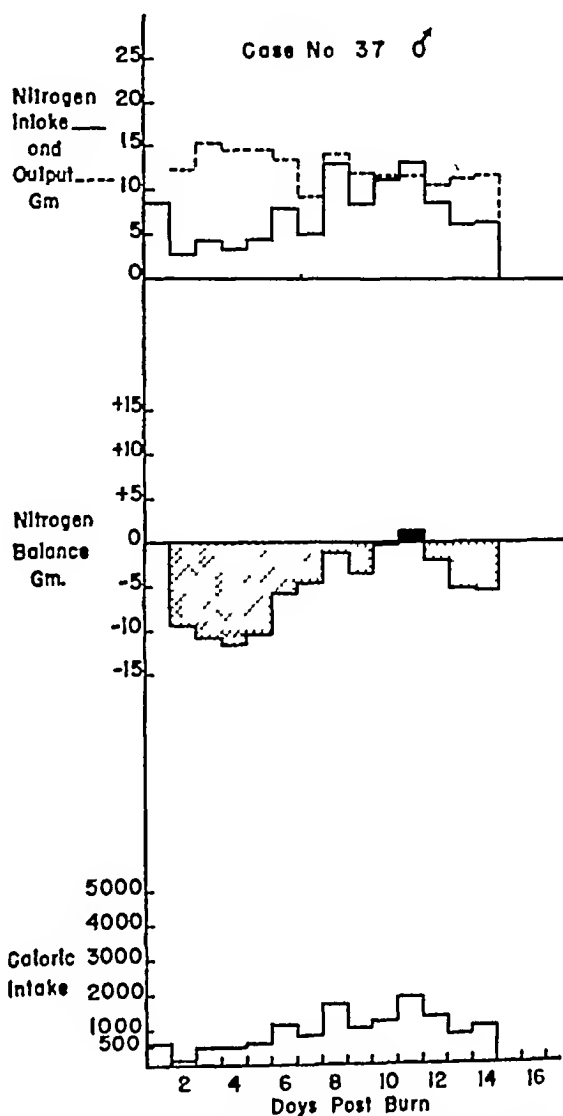


FIG 49—Nitrogen balance and caloric intake of Case 37, a man with moderate pulmonary damage and mild burns. He received four units of plasma in the first 48 hours.

cortex does activate such a conversion. Whether overactivity of the adrenal is responsible in shock in the human being for an increased production of sugar is not yet established. The observed increased excretion of 17-ketosteroids through the kidney following a burn or other trauma is in keeping with the concept of increased activity of the adrenal cortex.<sup>3,5,7</sup> The relation of the 17-ketosteroids to protein and carbohydrate metabolism appears to us to be still unsettled. Testosterone, which is the secretion of the interstitial cells of the testis and the end-product of which is excreted in the urine as a 17-ketosteroid, causes a retention of nitrogen when injected into a castrated or a normal man or woman.<sup>8</sup> Such action on nitrogen balance is the reverse of a conversion of protein to glucose. If the adrenal cortex also secretes steroids which are excreted in the urine as 17-ketosteroids is the action of these adrenal steroids like that of testosterone?

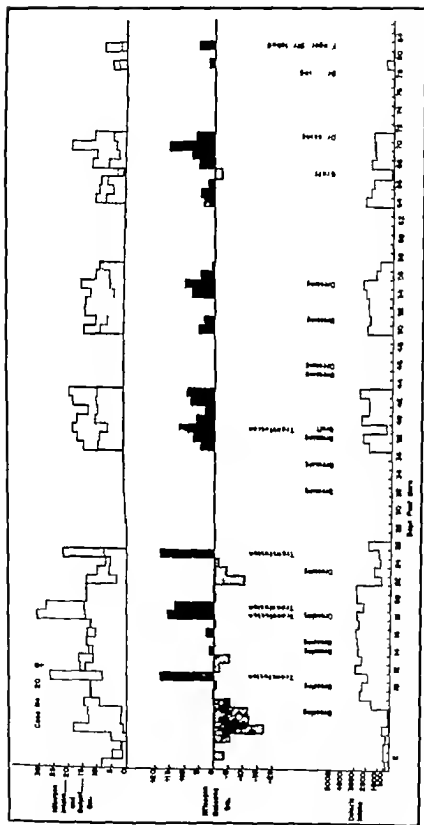


FIG. 50.—Nitrogen balance and caloric intake of Case 20 a woman with severe pulmonary damage and extensive burns. The caloric intake was increased on the eighth day by the introduction of a nasogastric tube for feeding. Patient had a tracheostomy. The peaks in nitrogen intake were caused by a side blood transfusion.

It is probable that the cortex of the adrenal is also a source of 17-ketosteroids. This is based on several clinical observations. First, these steroids are found in the urine of both man and woman, there is more in man than woman, but the difference disappears if the man is castrated.<sup>9</sup> Second, there is a diminished excretion of these steroids in the urine of patients with Addison's disease, in women with this disease there may be little or none.<sup>10</sup> (In Addison's disease, spontaneous hypoglycemia is sometimes encountered indicating an inadequate conversion of noncarbohydrate substances to glucose.) Third, patients with a functioning tumor of the adrenal cortex excrete an increased amount of 17-ketosteroids. Removal of the tumor is followed by an abrupt drop in the amount excreted. (Some of the patients in the presence of the tumor have an elevated blood glucose with sugar in the urine, intimating an increased production of glucose from non-carbohydrate sources.)

The theories regarding 17-ketosteroids and the adrenal cortex have been further complicated by the suggestion that the cortex secretes two types of hormones. One is a testosterone-like hormone, or "N" hormone, which causes retention of nitrogen, while the other, the "S" hormone, causes conversion of protein to sugar and, therefore, a loss of nitrogen.<sup>1, 5, 11</sup> The "N" hormone is supposedly excreted in the urine as a 17-ketosteroid, while the "S" is not and is determined only on biologic assay. In the burned patient it is suggested that the adrenal cortex at first puts out an excess of both "N" and "S" hormones but later only the "S", further, that a subsequently diminished output of "N" hormone, recorded as a decreased 17-ketosteroid excretion, represents a phase deleterious to wound healing, and that injection of testosterone might be beneficial.<sup>5</sup>

Such conflicting theories suggest premises based upon inadequate observations. It is possible that the chemical test which identifies the 17-ketosteroids is measuring steroids with differing physiologic properties. Investigation is needed.

Another problem is that of the source of the increased nitrogen excreted following the burn. It has been held that the nitrogen comes from the burned cells. It might, however, come from the plasma protein or some other noncellular storehouse of nitrogen. In the previous article on the care of shock, the possibility is discussed that the plasma proteins, which have seeped out into the extracellular spaces of the burned area, are excreted by the kidney when resorbed through the lymphatics into the general circulation. It is possible that these proteins are in some way denatured and are, therefore, no longer utilizable by the body when resorbed. Since the protein concentration of the edema fluid of the burned area is high owing to the abnormal capillary permeability induced by the burn, the excretion of such protein would mean a considerable loss of nitrogen.

If the increased nitrogen excreted comes from cells, there should be a parallel excretion of other substances contained in cells such as potassium and phosphate. If it comes from plasma protein, these cellular parallels

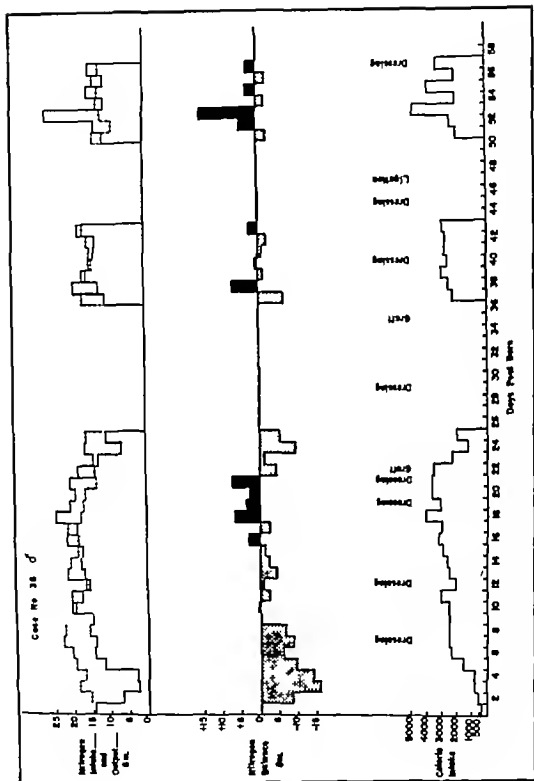


FIG. 11.—Nitrogen balance and caloric intake of Case 36, a man with moderate pulmonary damage and severe burns of head and hands. Although less injured than Case 20 (Fig. 50) or Case 13 (Fig. 6) his nitrogen excretion was greater.

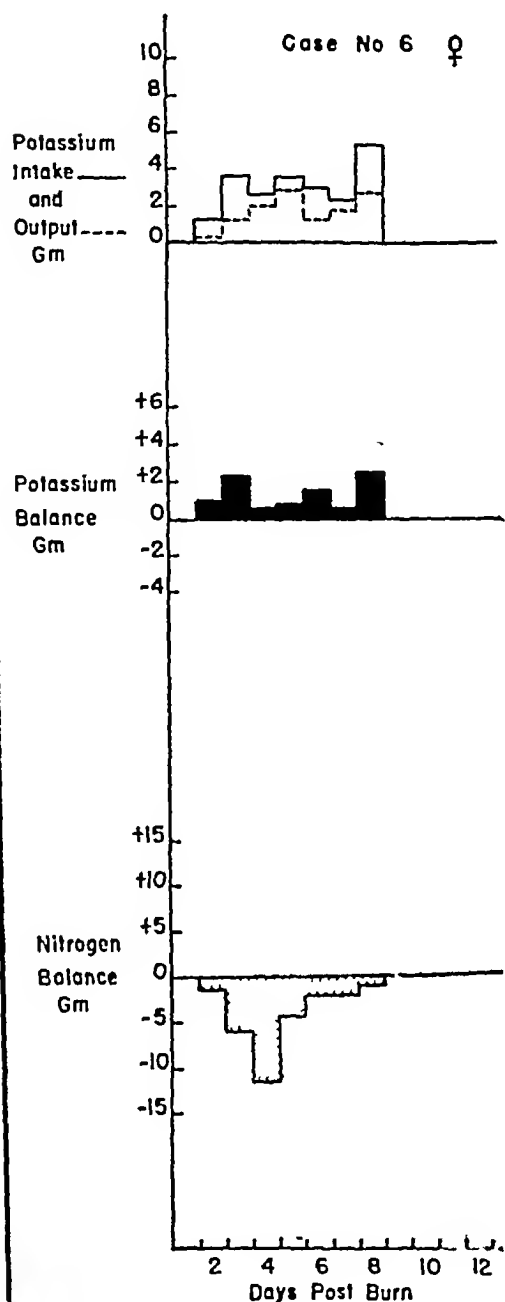


FIG. 52—Potassium balance, with nitrogen balance curve for comparison, in Case 6 (same case as Figures 48 and 57)

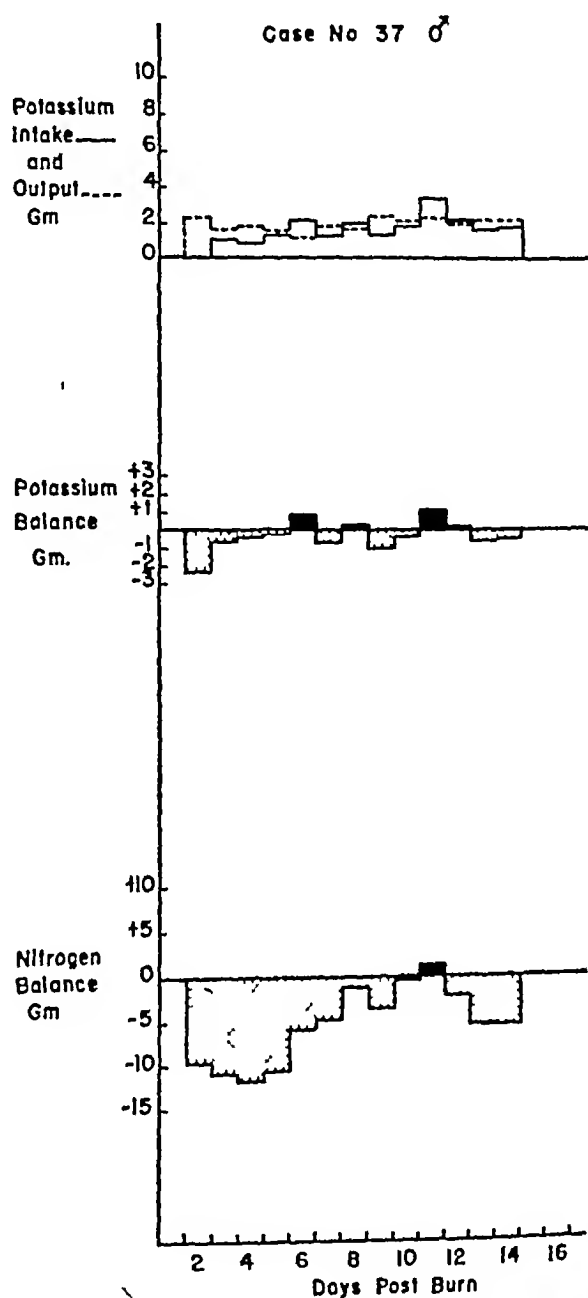


FIG. 53—Potassium balance with nitrogen balance curve for comparison, in Case 37 (same case as Figures 49 and 58)

would be missing. As far as we are aware, no studies of potassium and phosphate balance have been made in patients following burns.

In view of the conflicting evidence regarding the adrenal cortex and shock, it was decided to study in human burn shock the relations of 17-ketosteroid excretion to nitrogen metabolism, and of nitrogen to cellular metabolism.

#### EXPERIMENTS

Various aspects of metabolism were studied in 29 of the 39 victims of the Coconut Grove fire treated at the Massachusetts General Hospital.

## METABOLIC OBSERVATIONS

Twenty four hour urine specimens were collected on all these patients. Some specimens were not complete particularly in the first 72 hours when a few patients were incontinent. At times urine was lost with the stool and an occasional specimen was inadvertently thrown away.

*Nitrogen Balance*—The nitrogen excretion in the urine was measured in the 20 patients throughout their stay in the hospital. An accurately recorded intake of food is available for only nine of these patients throughout their hospital stay and in these nine only therefore is a complete nitrogen balance available\*. In five more patients food intakes were adequately recorded over considerable periods and the nitrogen balance has been calculated for these periods. On the remaining 15 patients only the outputs are available.

Smaller negative balances were encountered than anticipated. Of the nine patients with complete balances two died within the first 72 hours one having been in slight positive the other in slight negative balance. Of the seven survivors with complete balances two were in positive balance from the beginning owing in part to transfusions. One of these was the most severely burned of the 39 patients (Case 13. See Fig 61). Twenty eight per cent of the body was third degree burn and required grafting another 28 per cent was second degree making a total of 56 per cent of the body surface being severely burned with an unknown quantity of first degree burn (see colored section Fig 11). The other patient (Case 32) was also in positive balance owing to multiple transfusions given early to relieve anoxia.

The remaining five patients with complete balances showed negative balances up to nine days. During this period of negative balance, the caloric intake of the patients was inadequate for maintenance. It is believed that the negative balance is in large part accounted for by the low caloric and low protein intakes. The nitrogen data of four of these patients together with the caloric intakes are shown in Figures 48-51. An increased intake of protein apparently did not alter the nitrogen excretion.

In the five patients with incomplete balances as well as in the 15 with nitrogen excretions only the pattern of urinary nitrogen excretion is the same as in those with the complete balances. (The nitrogen data of the final patient with complete and two with incomplete balances are shown in Figures 59, 60 and 63.) Of interest in all of the 29 patients in whom the nitrogen excretion was determined is the constancy of the level of

\* The balance figures are constructed on the basis of the calculated nitrogen intake by mouth and by vein, against the nitrogen excretion in the urine. No attempt was made to measure the loss of nitrogen from the wound, that is, by leakage of the protein rich fluid into the bandages. Nor was the increased nitrogen of certain stools measured. Ordinarily the stools contain approximately 10 per cent of the total nitrogen excreted, but in those containing blood there was of course, additional nitrogen lost. (Five of the nine patients had stools positive to guaiac on one to three occasions in the first two weeks. There was no gross bleeding.)



excretion. It is a little higher during the first week, generally, than in later weeks.

The level of nitrogen excretion was higher in the males than in the females. This is in keeping with the differences in size and caloric requirements of the sexes. The extent of the skin burns did not influence the level of nitrogen excretion in either sex. Thus, of the four males with complete nitrogen balances, Case 37, with minor burns, showed the lowest excretion, Case 11, with the most extensive burns, and Case 29, with moderate burns, were in the middle, and Case 36, also with moderate burns, was highest. Cases 29 and 36 had almost identical anatomic areas involved, but the burns of Case 36 showed more infective organisms, and the initial grafts failed, in part, to survive.

*Potassium Balance*—Potassium balances were measured in six of the seven surviving patients with the complete nitrogen balance (Figs 52-55). The patients were essentially in potassium balance throughout, but all exceptions should be mentioned. Case 37 (see Fig 6) on the second day was in negative balance. In the two patients whose potassium balance charts are not shown (Cases 13 and 32), there was a positive potassium balance which was presumably due to the whole blood transfusions which these patients received.

The absence of a negative potassium balance, with the one exception noted, suggests that the nitrogen excreted in excess of the intake in the early days did not occur as a result of cell destruction.

*Calcium and Phosphorus Excretion*—The excretion of calcium and phosphorus was measured in the urine of one patient (Case 36), (see Fig 56). In view of the calculated intakes of these two substances in the diet, the output of both is within the expected level of normal. The fecal calciums were not measured, so a complete balance was not determined. The injury, or the rest in bed, was without apparent effect on the urinary excretion of these substances.

*17-Ketosteroid Excretion*—The urinary excretion of 17-ketosteroids has been measured in 23 patients. The method of determination is described elsewhere<sup>12, 13</sup>. The determinations were continued throughout the hospital stay, and in some instances after discharge. During the first week, assays were carried out daily, and thereafter at intervals of two to three days.

The data of eight typical patients are presented in Figures 57-64. The selection of the patients for illustration was based primarily on the length of the study and the severity of the injury which they had received. All had nitrogen and six had potassium studies in addition.\*

\* Normal values for 17-ketosteroids in this laboratory range between 6 to 15 mg per 24 hours in females, whereas, the range in the male is from 10 to 20 mg. Values at either extreme are unusual in our experience. In the same normal individual the daily fluctuations are not more than 25 per cent from the average of consecutive assays, and frequently are much less. Hence marked variations in output are considered significant.

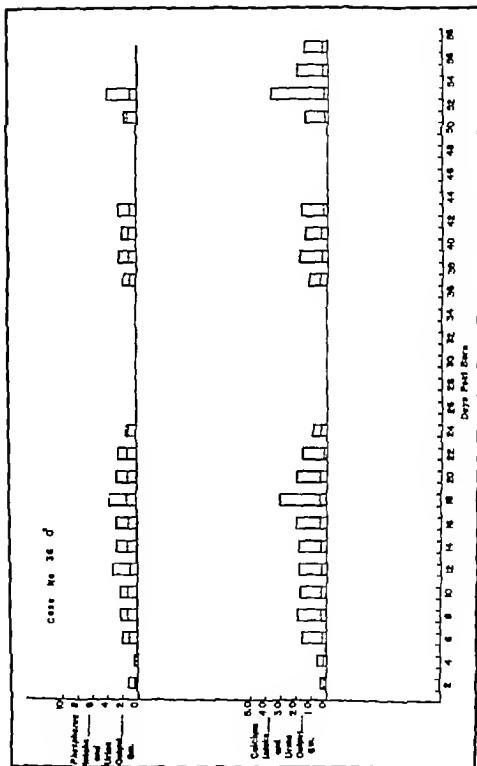


Fig. 16.—Calcium and phosphorus intake and urinary outputs in Case 36

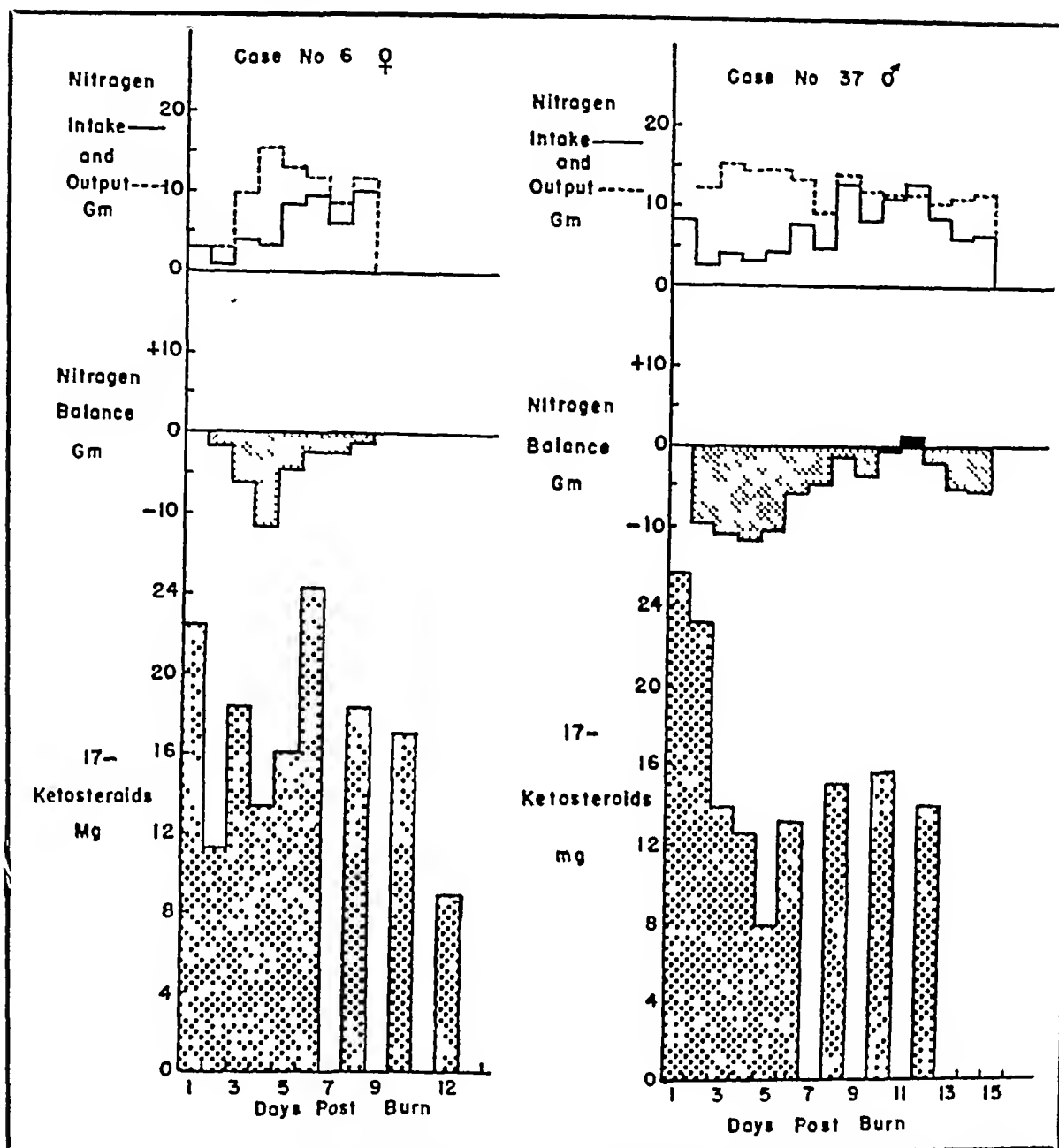


FIG. 57—17 ketosteroid excretion with nitrogen balance for comparison, in Case 6 (See also Figures 48 and 52.)

FIG. 58—17 ketosteroid excretion with nitrogen balance for comparison, in Case 37 (See also Figures 49 and 53.)

The levels of the 17-ketosteroid excretion are all high normal or above normal range for each sex during the first three to seven days after injury. Following this there is a precipitous drop to low normal or subnormal outputs. This decrease was found in all but a few patients who had the mildest injuries, or who were discharged from the hospital before such a drop could have been noted. Thereafter, except for occasional fluctuations, especially in the male patients, the 17-ketosteroid excretion remains at a relatively low level for a long period. In several, a rise to an average normal level did not occur until six weeks had elapsed (Figs. 63 and 64). In others this is not apparent until later (Figs. 60 and 62). In two women the levels are still low 113 days after the injury (Figs. 50 and 61). In general, the

duration of the low level of 17 ketosteroid excretion correlated closely with the extent of injury and period of convalescence.\*

#### CLINICAL CORRELATIONS

In the six female patients with third degree burns who remained in the hospital for grafting two observations were made of interest in view of the metabolic studies

The catamenia ceased in all six. It returned in one (Case 20) before discharge from the hospital at the end of two and one half months. In the next two it returned soon after discharge (Cases 23 and 28). In the remaining three (Cases 2, 8 and 13) it had not returned at four and one half months.

Increased growth of hair was noted in all six. In five the growth occurred on the face; in two there was a thick growth in one, on the cheek and in the other on the lips, chin and neck. In all six there was an increased growth on the extremities. The patients noticed this growth themselves and at first were hesitant to mention it. All were gratified to know it was not unexpected and that the others shared in it. With the return of catamenia in one patient the hair on the leg has already begun to drop out.

The pattern of 17-ketosteroid excretion was typical in all six patients. The increased hair growth occurred when the 17 ketosteroid excretion was low. The hair growth was thickest in two of the patients with the lowest and most prolonged depression of 17 ketosteroid excretion (Figs 59 and 61).

The 17 ketosteroid excretion rose to a normal level at about the time catamenia was resumed in the three patients in whom that has occurred. (One of these was Case 20 Fig 62)

#### COMMENT

The level of nitrogen excretion found in these patients was less than was anticipated and the ease with which nitrogen equilibrium was realized was a surprise. Even in these patients who were severely burned the loss of nitrogen was no greater than in those with relatively mild injury. The nitrogen balances observed no matter what the extent of the injury correlate more closely with the caloric and nitrogen intakes than with other factors. For example males with their higher caloric requirement excreted consistently larger amounts of nitrogen than the females.

The negative nitrogen balances observed during the periods of inadequate caloric and nitrogen intakes were not accompanied by a negative potassium

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\*It is to be noted that in Case 36 Figure 64, the levels are never below normal, but perusal of the data reveals the usual down swing with a typical valley before a rise to higher levels. Following discharge his levels are higher than average normal values. If these are normal for this individual then the lowest values are distinctly subnormal for him.

There was a more consistent daily excretion of 17-ketosteroids in the female than in the male. This may be attributed to additional gonadal activity in the male.

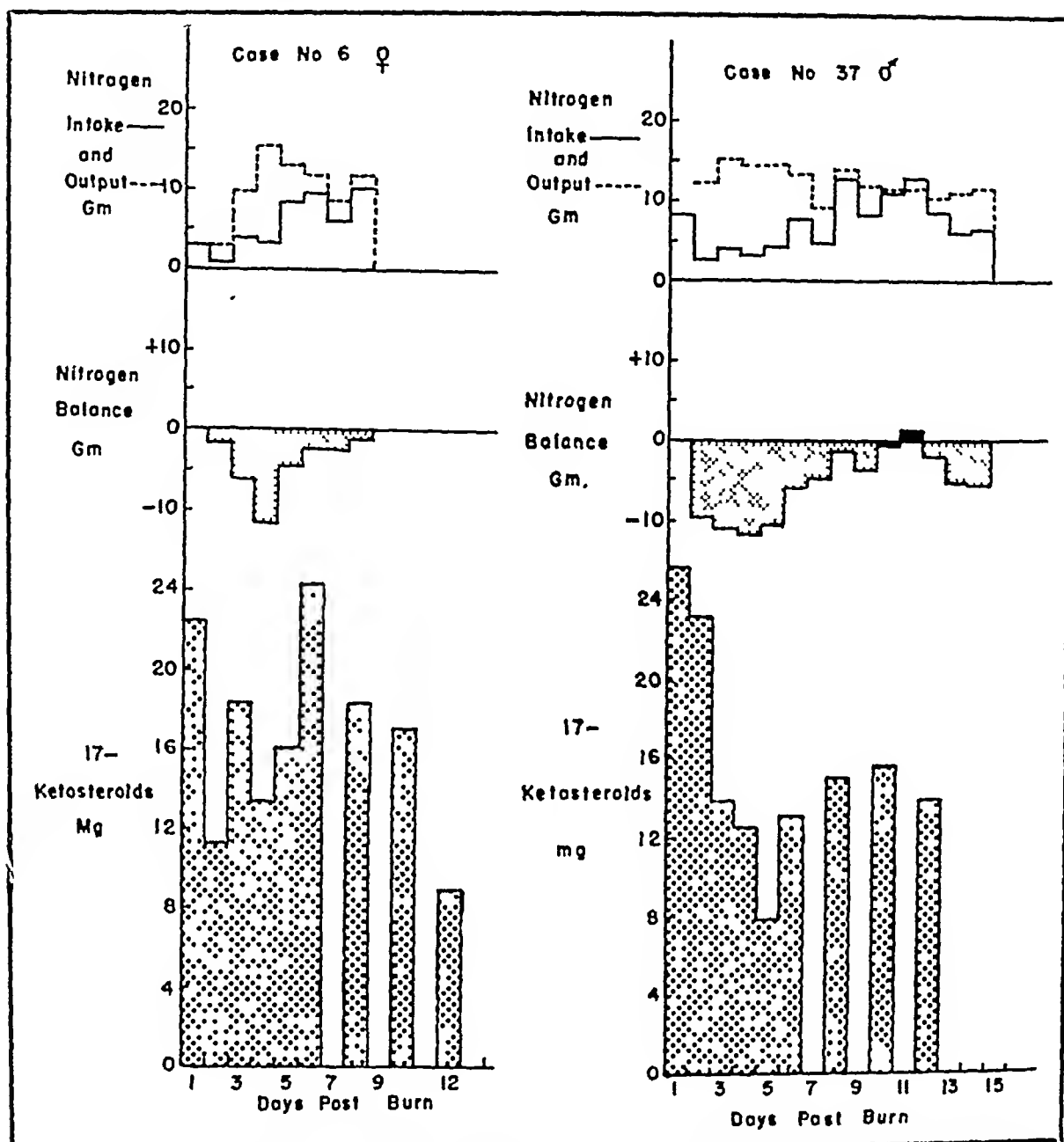


FIG. 57—17-ketosteroid excretion with nitrogen balance for comparison, in Case 6 (See also Figures 48 and 52.)

FIG. 58—17-ketosteroid excretion with nitrogen balance for comparison, in Case 37 (See also Figures 49 and 53.)

The levels of the 17-ketosteroid excretion are all high normal or above normal range for each sex during the first three to seven days after injury. Following this there is a precipitous drop to low normal or subnormal outputs. This decrease was found in all but a few patients who had the mildest injuries, or who were discharged from the hospital before such a drop could have been noted. Thereafter, except for occasional fluctuations, especially in the male patients, the 17-ketosteroid excretion remains at a relatively low level for a long period. In several, a rise to an average normal level did not occur until six weeks had elapsed (Figs. 63 and 64). In others this is not apparent until later (Figs. 60 and 62). In two women the levels are still low 113 days after the injury (Figs. 59 and 61). In general, the

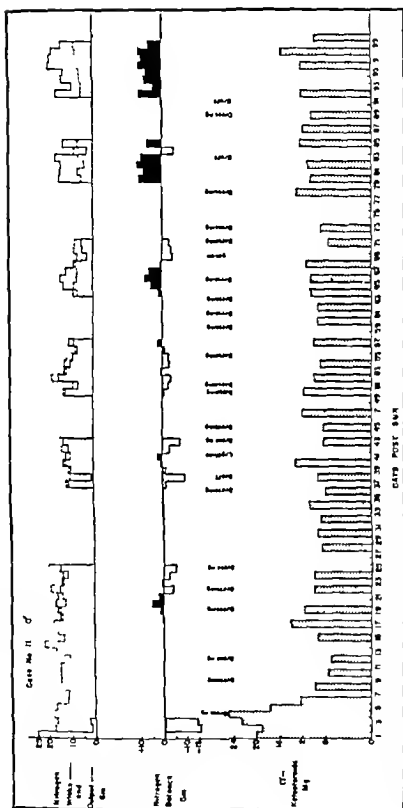
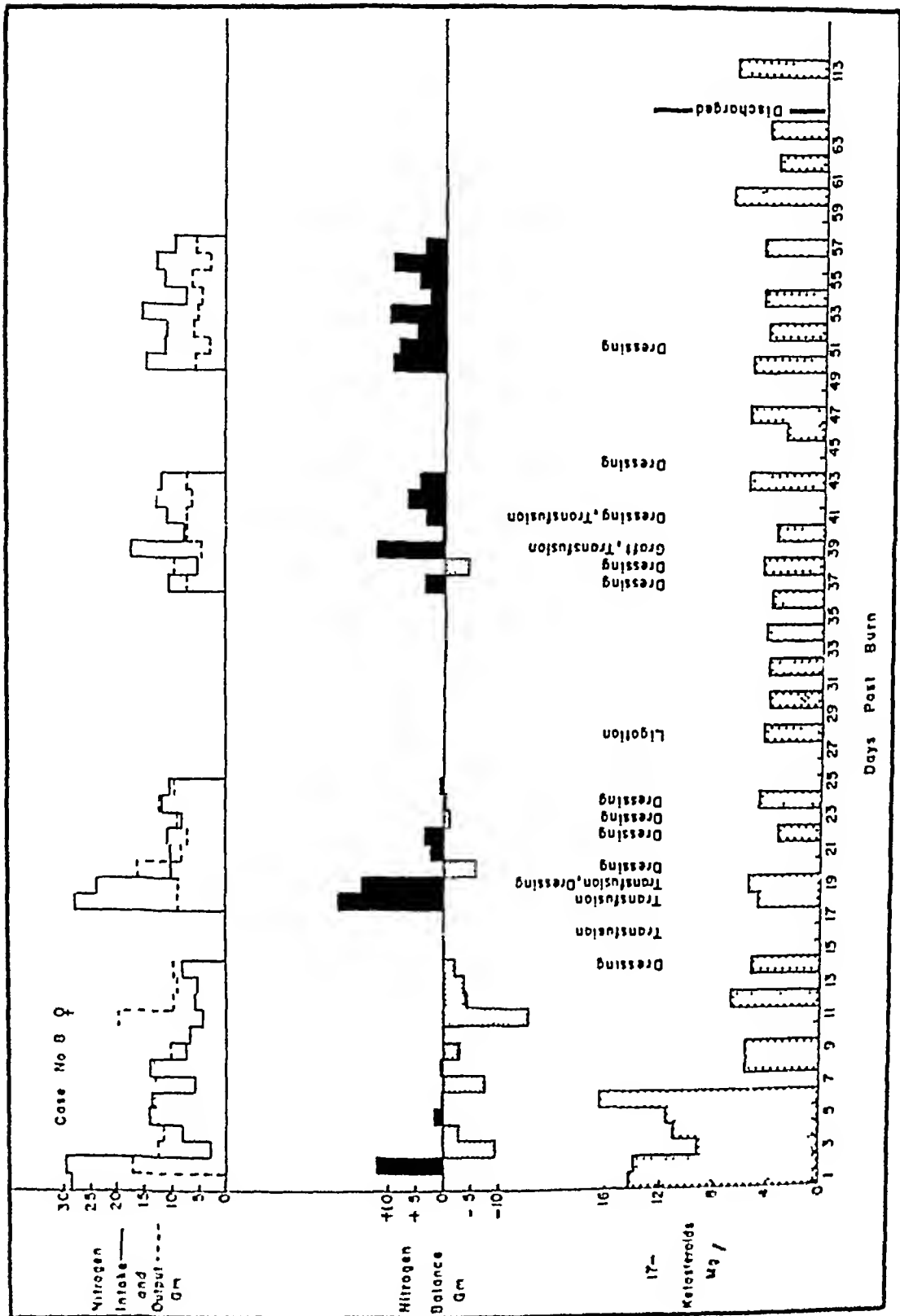


FIG. 60 — Nitrogen balance with nitrogen balance for comparison, in Case 11, a man with slight pulmonary emphysema and extensive burns. (The nitrogen excretion was lower than in Case 10, a man with lesser burns.)



with severe pulmonary damage and

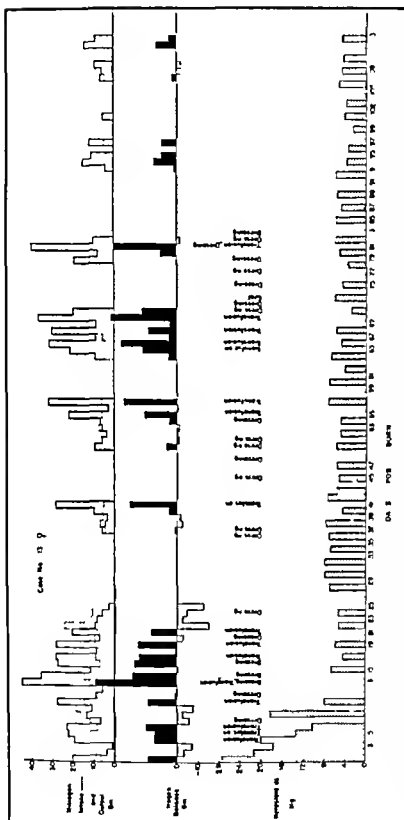


FIG. 61. — 7 ketosteroid excretion with nitrogen balance for comparison, in Case 13, a woman with moderate pulmonary damage and the most extensive burns. The peaks of nitrogen intake were the result of whole blood transfusions. Caloric intake was maintained for two periods with feedings through a nasal-gastric tube.





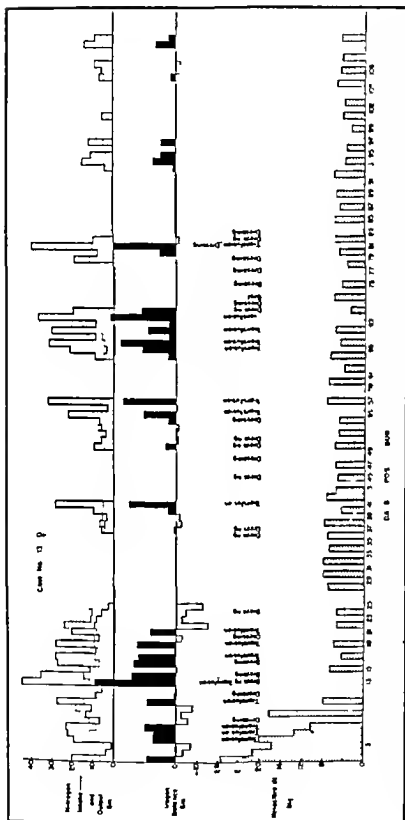


FIG. 61 — 7 ketosteroid excretion with nitrogen balance for comparison, in Case 13 a woman with moderate pulmonary damage and the most extensive berria. The peaks of nitrogen intake were the result of whole blood transfusions. Caloric intake was maintained for two periods with feedings through a nasal-gastric tube.

balance (with the exception of one day in a single patient) The nitrogen lost, therefore, was presumably not at the expense of cellular protein

These results are at some variance with what we had been led to expect from the observations of others<sup>2,3,4</sup> It is obvious that extensive deep burns are not necessarily accompanied by a large loss of nitrogen in the urine. We have, therefore, wondered what factors other than the burn itself might be involved. One thing which was clear, was the absence of invasive infection in the wounds of these patients. Infections such as typhoid or pneumonia cause a loss of nitrogen. At the moment, we ascribe the maintenance of the nitrogen balance of these patients to the relative lack of infection or, in other words, to the effectiveness of the chemotherapeutic program. Certainly, the simplified method of treating the burned surfaces did not lead to tissue destruction and protein loss. (See article on Surface Treatment)

A factor which has been reported to influence the nitrogen balance is adrenal cortical activity. It is well recognized that the cortex of the adrenal gland is disturbed following severe burns. A patient, studied recently at this hospital, following acid burns, died on the twenty-sixth day of extensive infection. The adrenal cortices were found at postmortem examination to have hypertrophied. On the other hand, acute adrenal cortical necrosis is known to occur, and was observed in two of these patients (Cases 7 and 27) who died on the third day. (See article on Pathology)

It is possible that the changes in 17-ketosteroid excretion observed in these patients reflect alterations in adrenal cortical function. The rise of excretion of these steroids in the first days after the burn would mean an increased activity and the subsequent decline, a depression. It has been suggested that the decline of 17-ketosteroid excretion after injury is due to preferential formation of the "S" hormone by the gland. An increased excretion of cortin-like (non-17-ketosteroid) hormone has been determined by the biologic method.<sup>5</sup> The decline in 17-ketosteroids could also be due to inability of the gland to secrete because of either anatomic or physiologic deficiency.

What emerges from the present studies is the constancy of the pattern of 17-ketosteroid excretion which follows this type of injury. In all patients in whom there was more than minimal damage, either in the form of pulmonary inflammation or surface burn, the excretion of 17-ketosteroids in the urine was initially at a high level almost certainly above normal to be followed by an abrupt decrease to a low level. This subnormal level remained until the patients had recovered.

This pattern does not follow all injuries or burns. For example, the 17-ketosteroid excretion of the patient mentioned who was found to have hypertrophy of the adrenal cortex following acid burns, showed normal levels throughout the 26 days of his survival. It is possible that other patterns will be found. It should also be pointed out that the urinary 17-ketosteroids represent only excretory products and that these may not be a direct index of secretion or utilization of the precursor. At the present time, all the e-

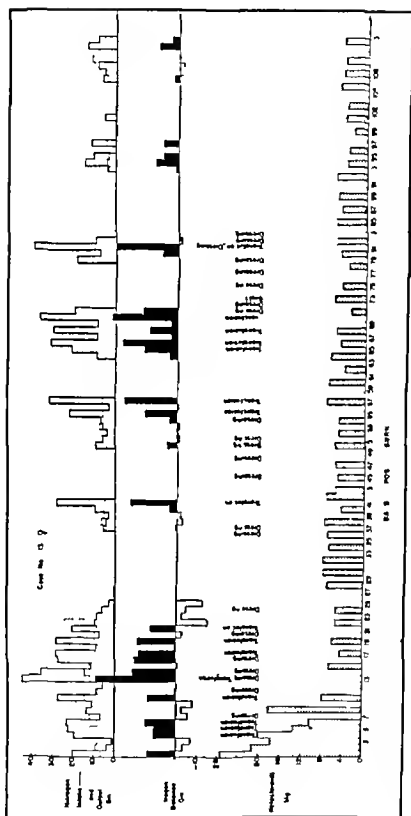
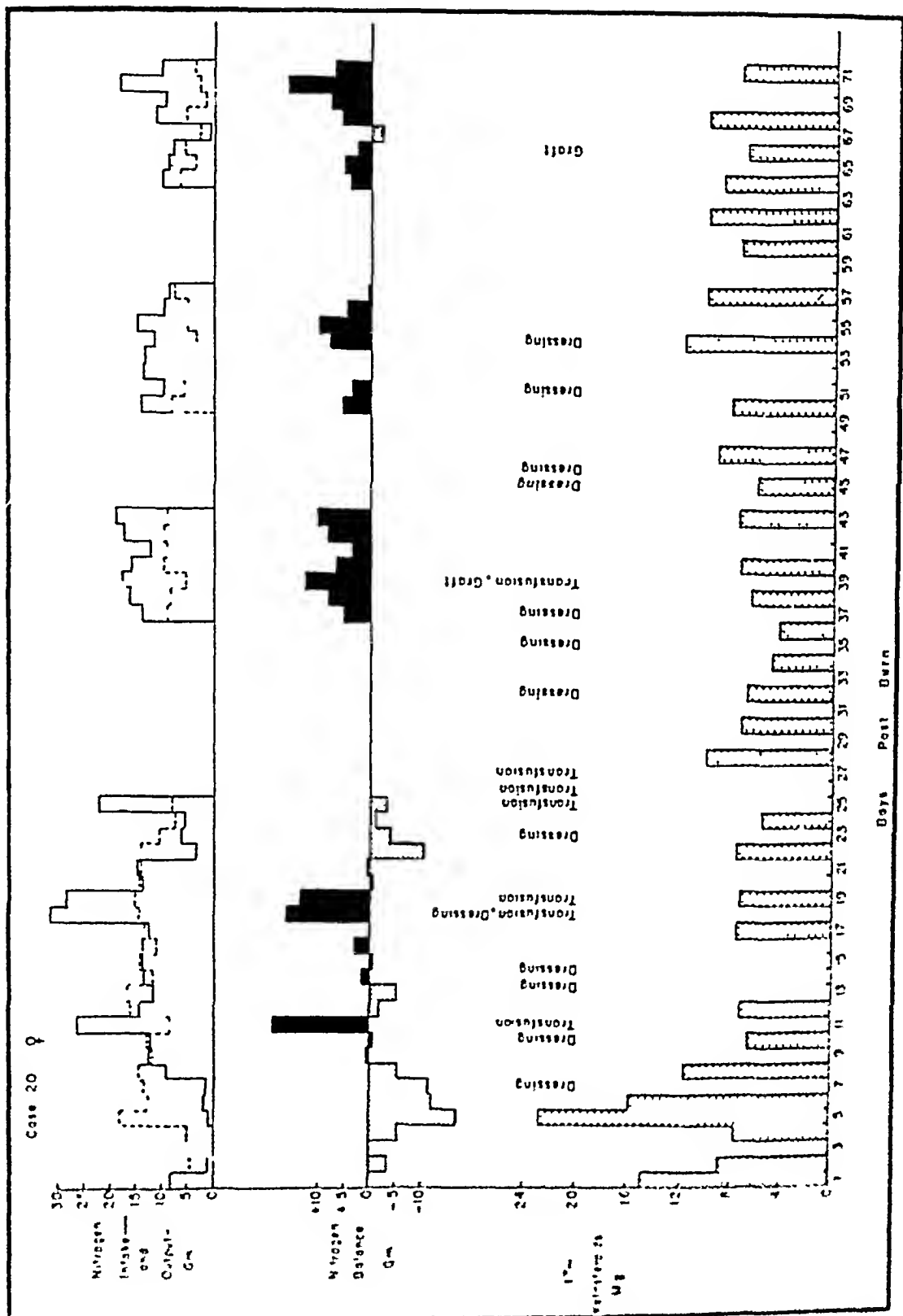


FIG. 6.—17 ketosteroid excretion, with nitrogen balance for comparison, in Case 15, a woman with moderate pulmonary damage and the most extensive burns. The peaks of nitrogen intake were the result of whole blood transfusions. Caloric intake was maintained for two periods with feedings through a nasal-gastric tube.



For further details on nitrogen balance see companion in Case 20 (see also Figures 51 and 52)

be said is that the relation of 17 ketosteroids to adrenal cortical function and the relation of both to nitrogen metabolism have not been settled.

Amenorrhea in the female patients was expected but hirsutism in the presence of a low 17 ketosteroid excretion was not. Ordinarily women with hirsutism have an excretion level at least in the upper limits of normal if not elevated as in those with true virilism due to tumor<sup>14</sup>. There are two possible explanations. Either utilization of masculinizing hormone was increased in these patients or the activity of the hormone present was uninhibited by a relatively low concentration of ovarian hormone. A situation similar to this latter possibility exists frequently after the spontaneous menopause or after ostration<sup>14</sup>.

No evidence was disclosed suggesting that the administration of testosterone would be beneficial to patients following burns *per se*. Nitrogen equilibrium was obtained readily without it even in the severely burned. The greater growth of abnormal hair which presumably would have occurred had testosterone been injected would have represented a further androgenic-estrogenic glandular imbalance. It is still theoretically possible that in patients depleted by prolonged sepsis and malnutrition with specific testicular and adrenal cortical insufficiencies testosterone therapy might be indicated. In like manner adrenal cortical therapy might prove useful.

#### SUMMARY AND CONCLUSIONS

Metabolic studies were carried out on 29 of the 39 victims of the Cocoa nut Grove disaster treated at the Massachusetts General Hospital. A complete nitrogen balance was obtained in nine patients and a potassium balance in six of these. The excretion of 17 ketosteroids in the urine was measured in 23 patients. In one the calcium and phosphorus excretion was determined.

In many patients in the first week there was a moderate negative nitrogen balance. For the same periods in these patients there was a caloric intake inadequate for maintenance. Nitrogen equilibrium was easily established when the caloric and nitrogen intakes were increased. The level of nitrogen excretion was fairly constant from day to day and was not altered by a rising protein intake; it was however less in later weeks than during the first week.

The level of nitrogen excretion bore no relation to the severity of the burn but males excreted more than females. It is believed that the moderate nitrogen loss encountered was due to the relative absence of invasive infection.

Potassium equilibrium was maintained. The source, therefore, of the nitrogen lost during the period of negative nitrogen balance was non-cellular protein.

The 17 ketosteroid excretion was elevated during a period corresponding to the negative nitrogen balance. After this first week it fell off abruptly to a low level. During this period the nitrogen excretion however did not change. The eventual return of the 17 ketosteroid excretion to normal was

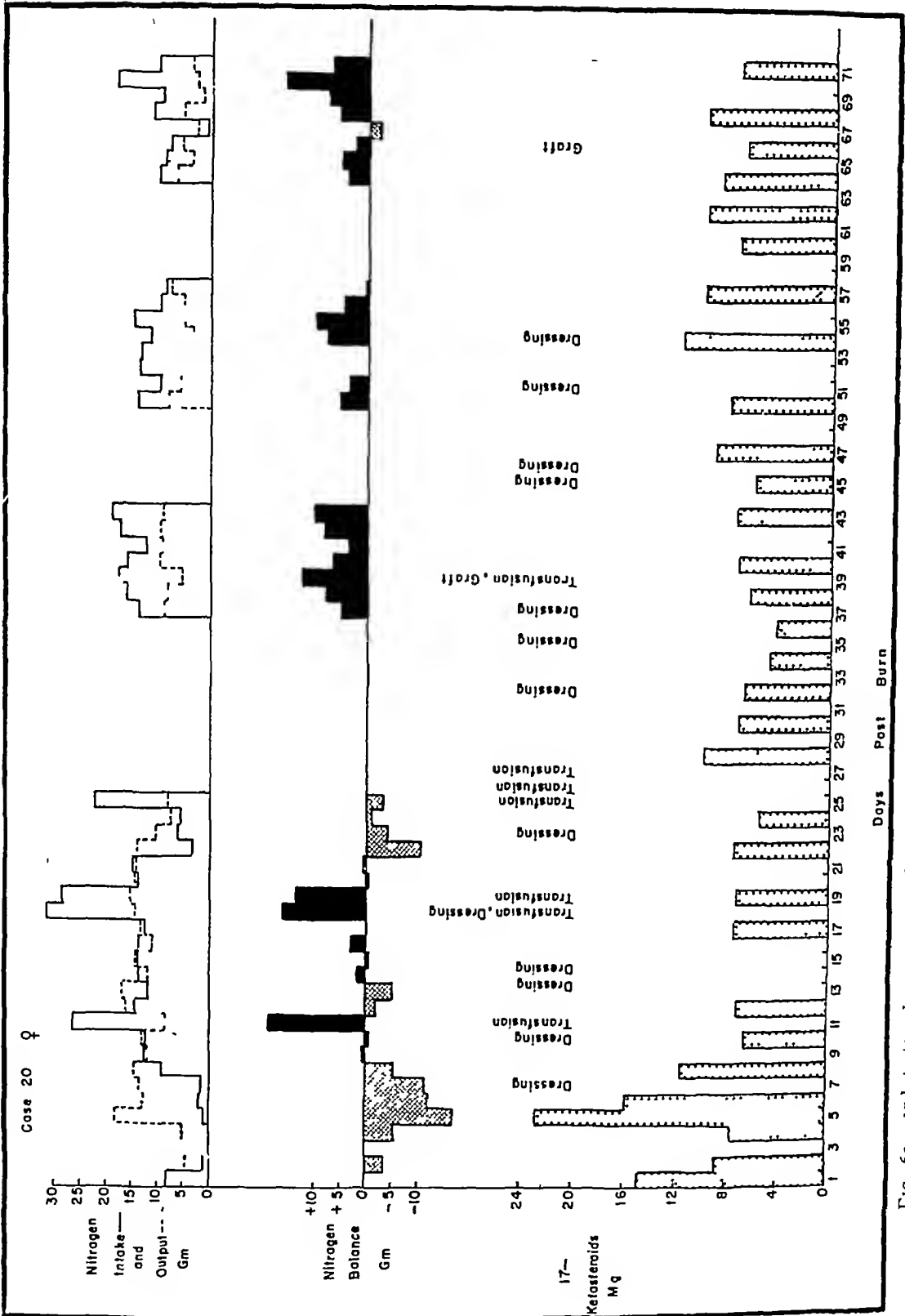


FIG 62 —17 ketosteroid excretion, with nitrogen balance for comparison, in Case 20 (See also Figures 50 and 54.)

be said is that the relation of 17 ketosteroids to adrenal cortical function, the relation of both to nitrogen metabolism have not been settled.

Amenorrhea in the female patients was expected but hirsutism and presence of a low 17 ketosteroid excretion was not. Ordinarily women with hirsutism have an excretion level at least in the upper limits of normal, if not elevated as in those with true virilism due to tumor<sup>11</sup>. There are two possible explanations. Either utilization of masculinizing hormone increased in these patients, or the activity of the hormone present was inhibited by a relatively low concentration of ovarian hormone. A situation similar to this latter possibility exists frequently after the spontaneous menopause or after castration<sup>12</sup>.

No evidence was disclosed suggesting that the administration of testosterone would be beneficial to patients following burns *per se*. Nitrogen equilibrium was obtained readily without it, even in the severely burned. Greater growth of abnormal hair, which presumably would have occurred if testosterone been injected, would have represented a further androgenic glandular imbalance. It is still theoretically possible that in patients depleted by prolonged sepsis and malnutrition, with specific testicular or adrenal cortical insufficiencies, testosterone therapy might be indicated. In like manner adrenal cortical therapy might prove useful.

#### SUMMARY AND CONCLUSIONS

Metabolic studies were carried out on 29 of the 39 victims of the Coconut Grove disaster treated at the Massachusetts General Hospital. A complete nitrogen balance was obtained in nine patients and a potassium balance in six of these. The excretion of 17 ketosteroids in the urine was measured in 23 patients. In one the calcium and phosphorus excretion was determined.

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Potassium equilibrium was maintained. The source, therefore, of the nitrogen lost during the period of negative nitrogen balance was cellular protein.

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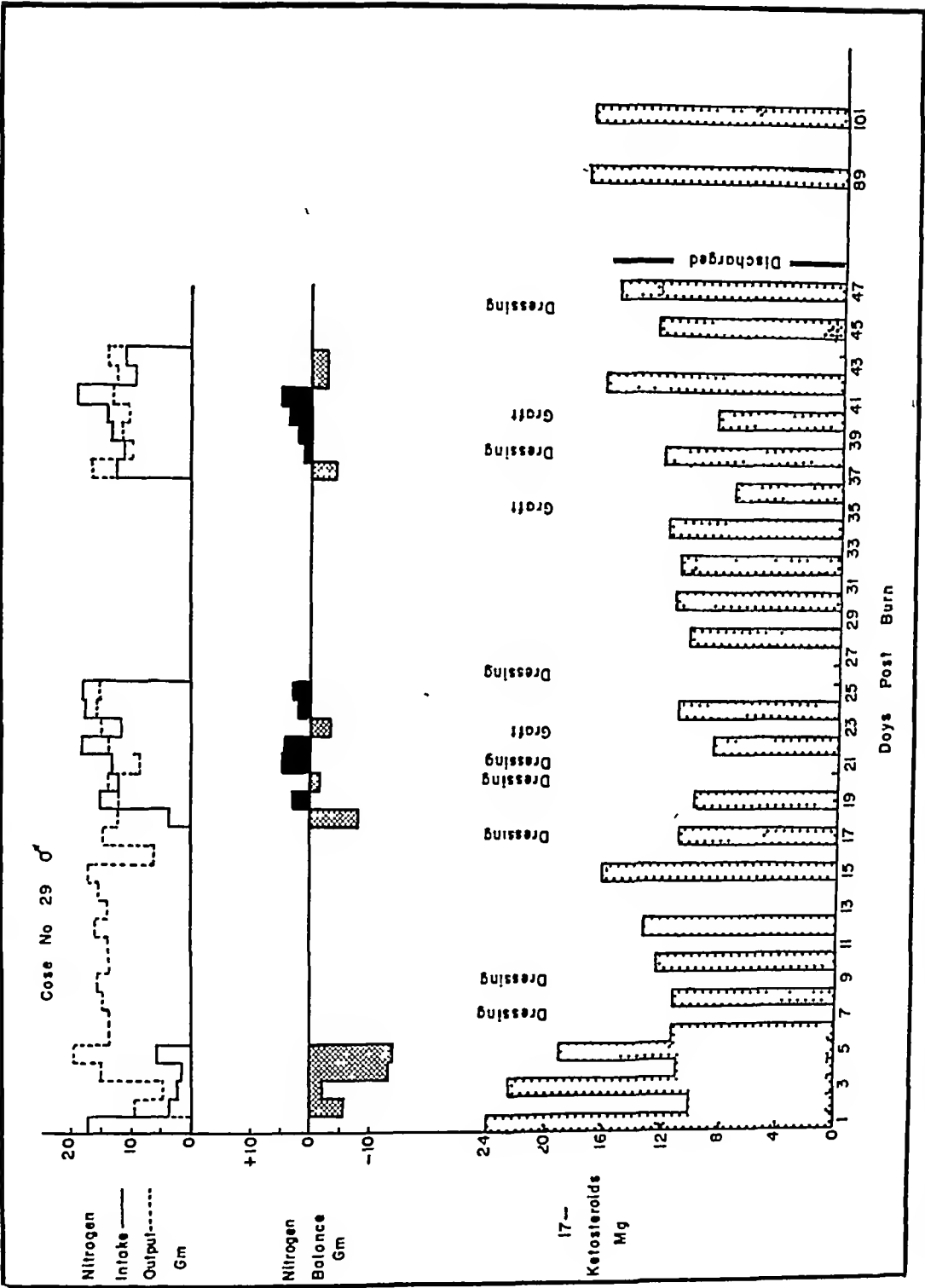


FIG 63—17 ketosteroid excretion, with nitrogen balance for comparison, in Case 29, a man with moderate pulmonary damage and severe burns of head and hands, comparable to Case 36

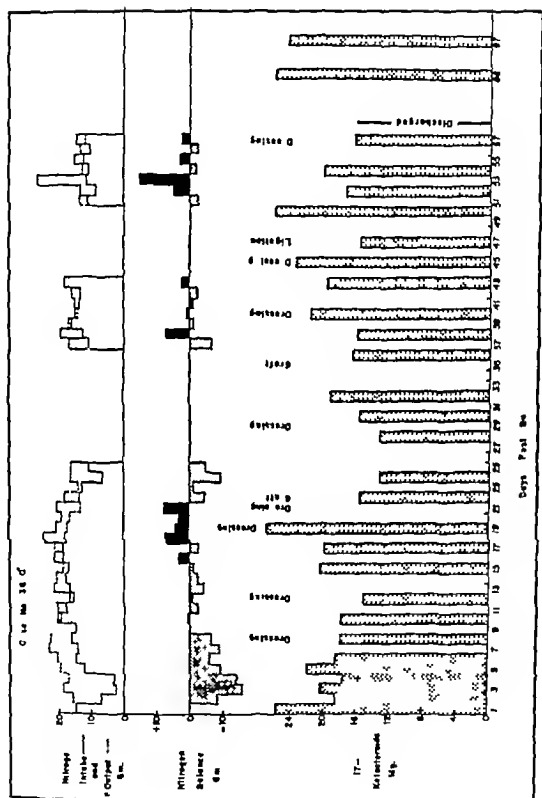


FIG. 64.— $\gamma$  Steroid excretion, with nitrogen balance for comparison in Case 36. (See also Figures 57, 58 and 56.)

simultaneous with the return to good health. The relation of the 17-ketosteroid excretion to nitrogen metabolism and to adrenal cortical activity was not settled.

An abnormal growth of hair was encountered in the six women hospitalized for more than three weeks, despite the low 17-ketosteroid excretion levels.

The ease with which nitrogen equilibrium was obtained, and the abnormal hair growth in the women, do not suggest testosterone as a routine therapy for patients following burns.

# REFERENCES

- 1 Selye, H. Studies on Adaptation. *Endocrinology*, **21**, 169, 1937.
- 2 Lucido, J. Metabolic and Blood Chemical Changes in a Severe Burn. *ANNALS OF SURGERY*, **111**, 640, 1940.
- 3 Browne, J. S. L. Personal communication. Conference on Bone and Wound Healing, December 11-12, 1942. Published by Josiah Macy, Jr., Foundation, New York, N. Y.
- 4 Taylor, F. H. L. Personal communication.
- 5 Albright, F. Personal communication. Conference on Bone and Wound Healing, December 11-12, 1942. Published by Josiah Macy, Jr., Foundation, New York, N. Y.
- 6 Long, C. N. H. A Discussion of the Mechanism of Action of Adrenal Cortical Hormones on Carbohydrate and Protein Metabolism. *Endocrinology*, **30**, 870, 1942.
- 7 Forbes, A. P. 17-Ketosteroid Excretion in Stress. Preliminary Report. Limited publication by Josiah Macy, Jr., Foundation, New York, N. Y., 1942.
- 8 Knowlton, K., Kenyon, A. T., Sandiford, I., Lotwin, G., Fricker, R. Comparative Study of Metabolic Effects of Estradiol Benzoate and Testosterone Propionate in Man. *J. Clin. Endocrinology*, **2**, 671, 1942.
- 9 Scott, W. W., and Vermeulen, C. Studies on Prostatic Cancer. Excretion of 17-Ketosteroids, Estrogens and Gonadotropins before and after Castration. *J. Clin. Endocrinology*, **2**, 450, 1942.
- 10 Fraser, R. W., Forbes, A. P., Albright, F., Sulkowitch, H., and Reitenstein, E. C., Jr. Colorimetric Assay of 17-Ketosteroids in Urine. *J. Clin. Endocrinology*, **1**, 234, 1941.
- 11 Albright, F., Smith, P. H., and Fraser, R. W. A Syndrome Characterized by Primary Ovarian Insufficiency and Decreased Stature. *Am. J. M. Sci.*, **204**, 625, 1942.
- 12 Nathanson, I. T., Towne, L. E., and Aub, J. C. Normal Excretion of Sex Hormones in Childhood. *Endocrinology*, **28**, 851, 1941.
- 13 Nathanson, I. T., and Wilson, H. Factors Affecting Colorimetric Urinary Ketosteroid Determinations. *Endocrinology*, in press.
- 14 Nathanson, I. T. Unpublished data.

## PROTOCOLS

The extent of the surface burns as recorded in the protocols is based on observations at the time of the first change of dressing (five to ten days after injury). Therefore the extent of the first degree burns could not be ascertained except of the face where there was obvious edema and no tissue destruction. The percentages are calculated on the basis of first second and third degree burns of the face, and of second and third degree burns of the rest of the body surface. The Berkav scale was used.

**Case 1—Male, age 7.** In trying to crawl out of the burning building the patient felt the flames about 2 up his back and over his face and believes that he breathed flames. He lost consciousness on reaching a floor.

**Condition on Arrival:** He was conscious but in shock, had slight cherry color of mucous membranes, and rales on auscultation of the chest.

**Extent of Burn:** First degree: Face. Second degree: Hands, back, thighs, legs. Third degree: Back. Total 15%. Corneal and Inhalation burns present.

**Plasma Therapy:** None. **Blood Pressure:** He maintained normal. **Laboratory Data:** Maximum hematocrit was 42% at 3 hours. Blood potassium 3.8 mEq. on second day; all other determinations also normal.

**Clinical Course:** The burned area did not require grafting. **Pulmonary Complication:** Patient developed moderate respiratory difficulty with some cough and expectoration. Chest roentgenograms, however, were negative and the respiratory difficulty cleared before discharge. **Subsequent Trauma:** None.

**Chemotherapy:** Sulfadiazine was continued until the twenty-sixth day. Penicillin: None. **Operation:** None.

**Discharge:** On the twenty-first day with one small grafting area on back and one on leg; subsequently healed. No respiratory difficulty.

**Case 2—Female, age 35.** Patient was in lobby as man rushed up stairs with clothes on fire. She was 6 feet from revolving door at entrance as he swept toward it by rush. Remembers choking lost consciousness at revolving door.

**Condition on Arrival:** Patient was hysterical, male and a great pain. She was not in shock. Mucous membranes were cherry-red color.

**Extent of Burns:** Second degree: Face, scalp, hand, thigh, leg. Third degree: Hand, wrist, scalp. Total 75%. Inhalation burns. (See color section Figs 13 and 14).

**Plasma Therapy:** Two units in first 4 hours. **Blood Pressure:** Seventy systolic soon after admission, gradually rose during the first 4 hours. **Laboratory Data:** Maximum hematocrit was 53% at 17 hours. Had sulfadiazine level of 7.4 mg. 4 hours after burn. Slowly developed an anoxia with hematocrit 134% on fifty-first day. Blood potassium was 3.4 mEq. on first day and 4.0 mEq. on second day. All other determinations normal.

**Clinical Course:** The burned areas were grafted with satisfactory results. Abnormal growth of hair occurred on face and forearms. Catamenia absent. **Pulmonary Complication:** Patient had received 45 gr. morphine subcutaneously on entry and was almost apneic on reaching the ward. Artificial respiration with oxygen through an intra-tracheal tube was carried on for five hours. Developed unicentric fibrillation which lasted for 24 hours, probable cause was anoxia. The patient was digitalized and oxygen therapy was continued for 6 days. The patient's respiratory difficulty with cough and some sputum continued for about 4 weeks gradually clearing.

Because of the possibility that the subsequent chest lesion were pulmonary infarcts, bilateral superficial femoral vein ligation was carried out on the fifty-second day. **Subsequent Trauma:** None.

**Chemotherapy:** Sulfadiazine continued until seventeenth day. Penicillin from the twentieth to the twenty-ninth day. **Operations:** Masti to left hand, 5. dominal flap skin graft to wrist on twenty-eighth day. Partial section of flap on forty-second day. Flap to left hand, abdominal flap detached on forty-eighth day. Bilateral superficial femoral vein ligation on fifty-second day. No clot found. Graft to abdominal wall defect on seventeenth day.

**Discharge:** On eighty-seventh day burns healed chest clear to Physical Therapy.

**Case 3—Male, age 23.** The patient was in the room in which the fire started, was unable to go upstairs because of the crowd so he crept in face with his hands raised and met the stairs until a fireman led him out of the building.

**Condition on Arrival:** He was conscious, in no shock, with no respiratory difficulty.

**Extent of Burns:** Slight first degree of nose and lips, slight inhalation.

**Plasma Therapy:** None. **Blood Pressure:** Normal.

**Clinical Course:** He was discharged to the Army Base the morning after the fire. **Pulmonary Complication:** Patient had a few rales in the right chest but no difficulty in breathing. **Subsequent Trauma:** None.

**Chemotherapy:** None. **Operations:** None. **Discharge:** First day condition good.

**Case 4—Female, age 27.** In the scramble the patient was knocked down, trampled upon and recalls inhaling smoke before she lost consciousness. She had not covered her face.

**Condition on Arrival:** She was conscious, in moderate shock, and coughing with rales at the left base.

**Extent of Burns:** Second degree: face. Total 25%. Corneal and Inhalation burns present.

**Plasma Therapy:** Three units in the first 24 hours. **Blood Pressure:** Remained essentially unchanged. **Laboratory Data:** Maximum hematocrit 53% at 63 hours. NPN on fourth day was 6 mg. All other determinations were normal.

**Clinical Course:** The burn of the skin healed without event. **Pulmonary Complication:** Patient continued to have moist, productive cough for several days. Lungs gradually cleared during the next two weeks by physical examination. **Subsequent Trauma:** None.

**Chemotherapy:** Sulfadiazine until the fifth day. Penicillin: None. **Operation:** None.

**Discharge:** On fourteenth day burns healed lungs clear.

**Case 5—Male, age 30.** Patient was overcome by dense smoke and lost consciousness; regained it only after admission to the hospital.

**Condition on Arrival:** He was unconscious but in no shock. He had considerable difficulty in breathing and was cyanotic with rales in both bases.

## PROTOCOLS

*Clinical Course* Burns healed uneventfully  
*Pulmonary Complication* Respiratory difficulty rapidly cleared  
*Subsequent Transfusions* None  
*Chemotherapy* Sulfadiazine continued until twelfth day  
*Penicillin* from sixth to eighth days  
*Operations* None

*Discharge* On twelfth day, burns of left hand clean but not healed, healed by six weeks

**Case 17—Female, age 43** In the confusion of the fire patient was knocked to the floor and was trampled by several people. She held her breath in order to avoid inhaling fumes, and was quickly led from the building by a fireman.

*Condition on Arrival* Conscious, no shock, normal breathing

*Extent of Burns* Second degree Face, hand, arm, shoulder Total 9% Inhalation burns (Face and hand debrided, cleansed and 5 per cent sulfathiazole ointment applied)

*Plasma Therapy* One unit on second day  
*Blood Pressure* Remained normal throughout  
*Laboratory Data* Maximum hematocrit was 52% at 37 hours Sulfadiazine level of bleb fluid was 2.2 mg on seventh day, simultaneous blood level was 4.6 mg Earlier sulfadiazine levels ranged from 9.1 mg at sixth hour to 4.0 mg at thirty-seventh hour

*Clinical Course—Pulmonary Complication* No respiratory symptoms but for the first two days had a few rales Lungs entirely clear on discharge  
*Subsequent Transfusions* None

*Chemotherapy* Sulfadiazine continued until twelfth day  
*Penicillin* None  
*Operations* None

*Discharge* On twelfth day, burns of arm clean but not healed, healed at two weeks

**Case 18—Female, age 42** Patient was in the balcony and tried to run from the flames when she lost consciousness. On coming to she was beneath a pile of bodies, and was pulled out by her legs.

*Condition on Arrival* Conscious, no shock, no respiratory difficulty

*Extent of Burns* First and second degree Face and hand Total 3.5% Slight inhalation burns

*Plasma Therapy* One unit in first 24 hours  
*Blood Pressure* Normal throughout  
*Laboratory Data* Maximum hematocrit was 46% at 8 hours

*Clinical Course* Burned areas remained clean  
*Pulmonary Complication* Patient developed slight productive cough which gradually decreased

Chest examination was normal on discharge  
*Subsequent Transfusions* None

*Chemotherapy* None  
*Operations* None

*Discharge* On sixth day, burns clean, healed

**Case 19—Male, age 42** Patient was in the main balcony, saw a flash of flames and thick smoke. He made his way downstairs with his fingers to his nostrils, and was pushed ahead by the throng. He remembers only taking one or two full breaths. Thereafter he recalls nothing.

*Condition on Arrival* Conscious, evidence of smoke inhalation

*Extent of Burns* Second degree Face, hands, scalp Total 5.5% Severe inhalation burns

*Plasma Therapy* Four units in first day  
*Blood Pressure* Remained normal  
*Laboratory Data* Maximum hematocrit 59% at 37 hours

Blood sulfadiazine level ranged from 5.5 mg down to zero on seventh day on which day bleb fluid level was 1.0 mg

*Clinical Course* The burns were a minor problem  
*Pulmonary Complication* Developed wheezing and other chest sounds a few hours after admission

Developed into severe bronchial asthma. Roentgenograms showed lobar emphysema with small areas of atelectasis on first day.

Treated with adrenalin and aminophylline. Gradually improved. Chest roentgenogram still showed evidence of some trapped air in the eighteenth week.

*Subsequent Transfusions* None

*Chemotherapy* Sulfadiazine for first eight days  
*Penicillin* sixth through twelfth days

*Operations* Attempted tracheotomy on fourth day, not accomplished because of massive edema of neck.

*Discharge* On thirty-second day, healed

**Case 20—Female, age 38** Patient's history was not obtained.

*Condition on Arrival* Shock impending, cyanotic

*Extent of Burns* Second degree Face, neck, back, scalp, arms, hands Third degree Arms, hands, shoulders Total 26.6% (see color section Fig 10) Inhalation burn

*Plasma Therapy* Three units on first day  
*Blood Pressure* Above 100 systolic throughout

*Laboratory Data* Maximum hematocrit 55% at 6 hours Blood potassium 3.3 and 3.7 mEq on first day Bleb fluid sulfadiazine 10.0 mg, with blood level of 10.6 mg on seventh day Metabolic observations recorded in article

*Clinical Course* General condition satisfactory. Required nasal gastric feedings because of burns of pharynx.

Abnormal growth of hair occurred on the lips, chin, and extremities. Absent catamenia.

*Pulmonary Complication* Respirations labored with productive cough. Respiratory embarrassment increased, with tracheal rattle.

Laryngoscopy showed edema of cords. On constant oxygen therapy.

Tracheotomy performed. Patient became comatose but rallied gradually.

Chest roentgenogram. See article by Schatzki.

*Subsequent Transfusions* Five of whole blood

*Chemotherapy* Sulfadiazine for first 24 days, leading to agranulocytosis (see article by Lyons).

Penicillin from sixth through fortieth days.

*Operations* Tracheotomy on third day. Split thickness skin grafts to arms and hands on thirty-ninth day.

Split thickness skin grafts to upper arms on sixty-seventh day.

*Discharge* On eighty-fourth day, healed except for proximal interphalangeal joint of left index finger, tracheotomy wound healed.

**Case 21—Female, age 26** The patient was not burned but inhaled a considerable amount of smoke for 25 minutes. On reaching the street she expectorated a great deal of "black stuff."

*Condition on Arrival* Conscious, in no shock, coughing

*Extent of Burn* None

*Plasma Therapy* None  
*Blood Pressure* Normal  
*Laboratory Data* None

*Clinical Course—Pulmonary Complication* Respiratory symptoms disappeared in 18 hours

*Chemotherapy* None  
*Operations* None

*Discharge* On first day, condition good

**Case 22—Male, age 39** Patient's history was not obtained.

*Condition on Arrival* In moderate shock, considerable difficulty in respiration. Cherry red color of denuded burned surfaces. Mucous membranes were charred and sooty.

*Extent of Burns* Second degree Face, scalp, neck, hands Total 11% Severe inhalation burns

*Plasma Therapy* Five units in 24 hours  
*Blood Pressure* Remained normal

*Laboratory Data* Maximum hematocrit 56% at 3 hours, 52% at 5 hours

*Clinical Course—Pulmonary Complication* For marked upper respiratory obstruction, tracheotomy had to be performed, at about 6 hours.

Respiratory distress apparently more marked in expiration, continued despite adrenalin, aminophylline and oxygen therapy.

Patient slowly failed and died 24 hours after original burn.

*Subsequent Transfusion* None

*Chemotherapy* Sulfadiazine continued until death  
*Penicillin* None  
*Operation* Tracheotomy on first day

*Discharge* On first day, dead

**Case 23—Female, age 30** Patient was seated in Melody Lounge when fire started. She ran upstairs and across dance floor, fell and remained prostrate near an exit.

The smoke was hot but she did not inhale much, did not cover her face. She did not lose consciousness.

She was wearing a heavy coat with a fur collar. She was dragged to safety.

*Condition on Arrival* Shock impending, no respiratory difficulty

*Extent of Burns* Second degree Face, neck,

hands, knee Third degree neck hands. Total 1% Slight inhalation burns.

*Plasma Therapy* Four units on first day. *Blood Pressure* Did not go below 14/60. *Laboratory Data* Maximum hematocrit 61% at 17 hours. Secondary anemia with hematocrit of 34% and plasma protein 5.5 Gm. on thirty-ninth day.

*Clinical Course* Transitory mental confusion with restlessness. Abnormal growth of hair occurred on face, forearm and legs. There has been a patchy loss of the hair since return of catamenia. *Primary Complication* Bilateral chest signs, chest roentgenogram remained negative. Vital capacity was 75% on seventh day. *Subsequent Transfusions* Three 1 whole blood.

*Chemotherapy* Sulfadiazine for first 12 days developed drug fever. Penicillin 1 through fourteenth day and until twenty-first through twenty-ninth day. *Operation* Skin graft to both hand on thirty-seventh day, complete 1 us of both grafts. Skin graft to both hands and wrist on fifty-second day; partial take. Skin graft to right hand on sixty-ninth day.

*Discharge* On eighty-third day healed, to Physical Therapy.

Case 24—Male age 29. Patient remembers no details.

*Condition on Arrival* Conscious in no shock, slightly cyanotic.

*Extent of Burns* First and second degree Face neck nostril tongue. Total 2%.

*Plasma Therapy* None. *Blood Pressure* Remained normal. *Laboratory Data* None.

*Clinical Course* Patient discharged approximately 1 hour following the fire with no complaints except difficulty in swallowing.

*Chemotherapy* None. *Operation* None. *Discharge* On first day to Fort Hays Hospital, condition apparently good.

Case 25—Male, age 46. History obtained.

*Condition on Arrival* Extremely hyperactive. Respiratory embarrassment required intermittent oxygen. Shock imparted on Cherry-red color of detached burned areas mucous membranes charred and sooty.

*Extent of Burns* Second and third degree Face, scalp hands, arms back. Estimated total 8% Severe inhalation burns. Corneal burns.

*Plasma Therapy* Five units on first day one on second. *Blood Pressure* Remained normal. *Laboratory Data* Maximum hematocrit 47% at 3 hours, dropped to 45% at 17 hours. Terminal rise in BUN to 64 mg., terminal blood potassium 4.3 mEq.

*Clinical Course* Primary Complication Restlessness became extreme. Respiratory embarrassment increased, required constant oxygen therapy. Marked hoarseness, nasal wallow Roentgenogram. See article by Schatzki. Paradoxical pulse developed.

*Chemotherapy* Sulfadiazine for two days. *Operations* None.

*Discharge* On second day dead. (Autopsy See article by Mallory and Brickley.)

Case 26—Male age 36. Patient was sitting at a table directly in front of the stage. On seeing the fire he headed toward the kitchen in the rear. He went down a short flight of stairs and found himself outdoors. He then reentered to search for the rest of his party and following this remembered nothing until his arrival at the hospital. Patient covered his face with a water-soaked napkin and tried to keep close to the floor.

*Condition on Arrival* Fainted with shock imparted by respiratory embarrassment. Shivering.

*Extent of Burns* Second degree Face hands. Total 5% Inhalation burns. Corneal burns.

*Plasma Therapy* One unit on first day. *Blood Pressure* 80 systolic on arrival rose in one-half hour to normal and remained so. *Laboratory Data* Maximum hematocrit 5% at 17 hours. Blood potassium 4.3 mEq. on both first and second days. All other determinations also normal.

*Clinical Course* General condition remained

good. *Inhalation Complication* Lungs remained clear until second day when some rales developed. Roentgenograms of the chest on the first and second days were negative. On the third day some atelectasis and trapped air were seen and were still present on the eighteenth day. Vital capacity 50% on tenth day. *Subsequent Transfusions* None.

*Chemotherapy* Sulfadiazine for first 1 days. Drug fever and rash developed. *Operations* None.

*Discharge* On twenty-first day chest clear and burn completely healed.

Case 27—Female age 18. History was not obtained.

*Condition on Arrival* Manic and hysterical in moderate shock, considerable difficulty with respiration.

*Extent of Burns* Second and third degree Face, arms, back, buttocks, hands. Total 22% Inhalation burn.

*Plasma Therapy* Eight units in first 24 hours five units second and third days. *Blood Pressure* Dropped to 80/60 soon after admission gradually rose. *Laboratory Data* Maximum hematocrit was 56% at 3 and 15 hours (see Fig 47 article on Shock).

*Clinical Course* Acute dilatation of the stomach was relieved by aspiration with a catheter. Adrenal cortical extract was given. *Primary Complication* Continued to have respiratory difficulty, requiring artificial respiration and oxygen administration. Aminophylline was given for the bronchiolar obstruction without effect. Developed paradoxical pulse. She rapidly failed and on the third day died of pulmonary failure. *Subsequent Transfusions* None.

*Chemotherapy* Sulfadiazine continued until death. *Operation* None.

*Discharge* On third day dead. (Autopsy See article by Mallory and Brickley.)

Case 28—Female age 26. Patient was sitting in Melody Lounge; saw the palm tree catch on fire. When the fire spread, across the ceiling she tried to run to the stairs but the flames burned her back. She breathed in a lot of smoke and fumes, fainted and became unconscious.

*Condition on Arrival* Shock impending. II d minimal hoarseness.

*Extent of Burns* Second degree Face neck, back, hands, arms. Third degree Back, hand, arm. Total 34.5% Corneal burn. Inhalation burn (see color section Fig 16).

*Plasma Therapy* Eight units on first day two on second day one on third day. *Blood Pressure* Dropped to 80/55 on first morning, promptly returned to normal. *Laboratory Data* Maximum hematocrit was 64% at 11 hours. Blood potassium was 3.7 mEq. on second day. Bile fluid sulfadiazine was 6 mg. and blood level 6.6 mg. on fifth day; on the sixth day bile fluid level 6.0 mg. and seventh day blood level 4.0 mg.; on the twelfth day bile fluid 4.9 mg. with blood levels of 4.1 mg. on tenth day and only smallest possible trace on fourteenth day.

*Clinical Course* Abnormal growth of hair occurred on arms and legs, slight on face. Absent catamenia. *Primary Complication* Breath sounds somewhat diminished on left. Roentgenogram negative. *Subsequent Transfusions* One whole blood on sixteenth day.

*Chemotherapy* Sulfadiazine for first 1 days. Drug fever and rash developed. Penicillin given from sixth through twenty-eighth days. *Operation* Skin graft to hand and arm on fifty-first day.

*Discharge* On fifty-seventh day completely healed except for small spot on back subcutaneously healed. To Physical Therapy.

Case 29—Male age 40. Patient was in the new bar when the fire started. When on his way to the door saw a sheet of flame and smoke. He was pulled out by a fireman, fainted and recovered consciousness on arrival at the hospital.

## PROTOCOLS

*Condition on Arrival* Shock impending, few râles in the chest

*Extent of Burns* Second degree Face, ears, scalp, hands, arms Third degree Hands, arms, scalp Total 12.5% Inhalation burn (See color section, Fig 15)

*Plasma Therapy* Six units on first, and one on second days *Blood Pressure* Normal *Laboratory Data* Maximum hematocrit 61% at 10 hours, otherwise all determinations were normal

*Clinical Course-Pulmonary Complication* Small areas of atelectasis developed in the right chest as shown by roentgenogram from fourth to eleventh days Vital capacity 79% on seventh day, 135% on tenth day *Subsequent Transfusion* None

*Chemotherapy* Sulfadiazine for first 28 days Penicillin from twenty-second through twenty-eighth days *Operations* Skin graft to left hand on twenty-third day Skin graft to right hand on thirty-fifth day

*Discharge* On fortieth day, healed, to Physical Therapy

**Case 30—Female, age 24** Patient was in the main room opposite the stage, saw people running from the main entrance followed by thick clouds of smoke. She saw no flame. She escaped through the kitchen. The smoke was very bad. After having fallen to the floor she was dragged by her husband through a window. She lost consciousness partially.

*Condition on Arrival* Good, some evidence of smoke inhalation

*Extent of Burns* No surface burns Slight inhalation burns

*Plasma Therapy* None *Blood Pressure* Normal *Laboratory Data* Hematocrits normal Blood chloride level was slightly elevated for four days, otherwise all determinations were normal

*Clinical Course* Had nausea and vomiting, headache *Pulmonary Complication* Developed moist râles in chest. Chest remained clear by roentgenogram

*Chemotherapy* Sulfadiazine on first day only *Operation* None

*Discharge* On sixth day, condition good

**Case 31—Male, age 27** Patient was exposed to smoke for about 25 minutes while dragging his wife and other people out of the fire. He came unconscious and was carried across the street where he regained consciousness in a few minutes, took a taxi to the hospital.

*Condition on Arrival* Good, calm, no respiratory difficulty, covered with soot

*Extent of Burns* Second degree Lips, nares Total 0.5%

*Plasma Therapy* None *Blood Pressure* Normal *Laboratory Data* None

*Clinical Course* General condition remained excellent *Pulmonary Complication* Developed some coarse rhonchi, râles in the chest, and coughed up smoky mucus

*Chemotherapy* None *Operation* None.

*Discharge* On first day to Fort Banks Hospital

**Case 32—Female, age 35** History not obtained *Condition on Arrival* Profound shock, unconscious with stertorous breathing Cherry red color of burns and mucous membranes

*Extent of Burns* Second degree Face, hand, forearm Total 6% Severe inhalation burns

*Plasma Therapy* Six units on first day, two on second, and three on third *Blood Pressure* Returned to normal after first three units of plasma *Laboratory Data* Hematocrit was never above normal but varied between 29 and 40% for first four days, arterial blood oxygen content 10.0 vol % on third day, whole blood transfusions were given Plasma protein was never below 6.9 gm Blood potassium was 3.2 mEq on first day Prothrombin time was prolonged on fourth day Blood chloride was 121 mEq on second day Van den Bergh was 7.3 mg on seventh day

*Clinical Course* The burns were a minor problem. Was extremely restless, given paraldehyde

Pulse not obtainable Remained unresponsive and often had a senseless grin on her face. Psychiatric consultant felt that there was severe cerebral damage which was irreparable *Pulmonary Complication* Oxygen given immediately Cyanosis increased and tracheotomy was performed Roentgenogram showed areas of atelectasis from first to seventeenth days *Subsequent Transfusions* Two whole blood on third day, four whole blood later

*Chemotherapy* Sulfadiazine for first 13 days Penicillin sixth through twelfth days *Operation* Tracheotomy on second day

*Discharge* On sixty-seventh day, burns healed, tracheotomy wound healed, lungs clear, evidence of central nervous system damage present

**Case 33—Male, age 43** History not obtained *Condition on Arrival* Very poor, required oxygen

*Extent of Burns* Second and third degree Face, hands, chest, back, abdomen, legs Estimated total 50% Severe inhalation burns

*Plasma Therapy* Four units in first 12 hours *Blood Pressure* Normal *Laboratory Data* Hematocrit was 58% at 3 hours

*Clinical Course-Pulmonary Complication* Respiration and color remained poor Coarse moist râles were present throughout lungs Given coramine and aminophylline without avail

*Chemotherapy* Sulfadiazine once *Operation* None

*Discharge* 13 hours after injury, dead

**Case 34—Female, age 40** History not obtained

*Condition on Arrival* Profound shock, unconscious, respiratory difficulty requiring oxygen

*Extent of Burns* Third degree Face, neck, hands, back, legs, abdomen Estimated total 70% Severe inhalation burns

*Plasma Therapy* Eight units in first day *Blood Pressure* Not obtainable *Laboratory Data* For determinations see Figure 46 in article on Shock

*Clinical Course* Color remained poor Given adrenal cortical extract repeatedly Roentgenogram first morning showed some atelectasis and acute dilatation of the stomach Gastric aspiration carried out Small urinary output Condition remained poor

*Chemotherapy* Sulfadiazine first day *Operation* None

*Discharge* 26 hours after admission, dead

**Case 35—Female, age 45** Patient was seated 30 or 40 feet from the head of the stairs when she saw a flash of flame coming up the stairs. She ran in the opposite direction, covering her face with her hat. She remembers that the room was filled with choking smoke and then she lost consciousness.

*Condition on Arrival* Unconscious, in mild shock, breathing normally

*Extent of Burns* Second degree Face, hand Total 3% Corneal burns

*Plasma Therapy* One unit in first 24 hours *Blood Pressure* 74/55 on entry, rose to 90/60 in 8 hours and was thereafter normal *Laboratory Data* Maximum hematocrit was 43% at 17 hours Blood chloride was 116 mEq at 37 hours Other determinations were normal

*Clinical Course* Because of the death of her husband in the fire the patient developed marked mental disturbance. Although further hospitalization and psychiatric care were advised, patient left the hospital against advice *Pulmonary Complication* Mild respiratory difficulty cleared within a week

*Chemotherapy* Sulfadiazine continued until ninth day *Operation* None

*Discharge* On tenth day, burns healed with reactive depression

**Case 36—Male, age 28** Patient arrived on the floor. Heat and smoke troubled him most. He was rescued by firemen. He retained consciousness throughout.

## PROTOCOLS

*Condition on Arrival* Conscious breath sounds were diminished at the right base cyanotic.

*Extent of Burns* Second degree: Face scalp neck, ears hands. Third degree (hand) Total: 8.5% Corneal and inhalation burns.

*Plasma Therapy* Five units on first day two on second. *Blood Pressure* Normal. *Laboratory Data* Maximum hematocrit was 59% at 12 hours. Metabolic observations are given in article.

*Clinical Course* *Pulmonary Complication* Marked hoarseness with diminution of breath sounds. Small areas of atelectasis by roentgenogram from fourth to eleventh days. Vital capacity 70% on tenth day. *Subsequent Transfusions* One whole blood.

*Chemotherapy* Sulfadiazine for first 12 days. Penicillin with through thirtieth days. *Operations* Skin graft to both hand on twenty-fourth and thirty-fifth days. Left superficial femoral vein ligation on forty-seventh day home clot evacuated.

*Discharge* On fifty-eighth day burns healed, to Physical Therapy.

**Case 37**—Male age 32 Patient saw the room on fire suddenly reached for a coat from the wall and covered his face. While struggling in the crowd he lost consciousness.

*Condition on Arrival* Unconscious cold, tremor breathing deeply and steadily not in shock.

*Extent of Burns* Second degree: Face neck, hand. Total 3% Corneal and inhalation burns.

*Plasma Therapy* Three units in first 24 hours, one on second day. *Blood Pressure* Remained normal. *Laboratory Data* Maximum hematocrit was 56% at 37 hours. Blood potassium was 3.5 mEq. on first day. All other determinations were normal. Metabolic observations recorded in article.

*Clinical Course* Deceased of feelings of depression and an icy over the death of his wife he had to be transferred to psychiatric service for ten days before discharge. *Pulmonary Complication* Within few hours developed cyanosis cough, and hoarse rales in both lungs. Respiratory difficulty cleared during the next week. *Subsequent Transfusions* None.

*Chemotherapy* Sulfadiazine continued until sixteenth day. *Operation* None.

*Discharge* On seventeenth day burns healed, lungs normal.

**Case 38**—Male age 36 Patient was at the room bar saw smoke and flame coming up from downstairs. He was caught in mob and carried in the direction of the new cocktail room the smoke was severe. He was held down by the crowd and into a small room from which there

was no exit. He lay down for 15 to 30 minutes. When no fire reached him he got up and clambered over bodies lying about. He heard somebody about "Here's a man who I still alive." He was put in a taxi and brought to the hospital. He never lost consciousness.

*Condition on Arrival* Shock impending a respiratory difficulty.

*Extent of Burns* Second degree: Face, neck, scalp, back, hand, leg, foot. Third degree: Ankle. Total 21.5% Inhalation burns.

*Plasma Therapy* Six units on first day one on second one on third. *Blood Pressure* Remained normal. *Laboratory Data* Maximum hematocrit was 63% at 6 hours. Blood potassium was 4.1 mEq. and 4.5 mEq. on first day. 4 mEq. on second day. Urine fluid sulfadiazine level was 7.4 mg. on seventh day with simultaneous blood level of 6.4 mg. Blood level had been 9.5 mg. on fourth day. Had elevated NPN (see article on Shock).

*Clinical Course* Nausea and vomiting for all days. *Pulmonary Complication* No pulmonary symptom or signs. Roentgenogram negative on second day. Small areas of atelectasis and small amount of trapped air by roentgenogram from fourth to eleventh days. Vital capacity 62% on seventh day. *Subsequent Transfusions* None.

*Chemotherapy* Sulfadiazine for first 11 days. Penicillin from fifth through eleventh days. *Operations* None.

*Discharge* On fourteenth day to Chelsea Hospital chest clear burns unhealed.

**Case 39**—Female age 3 Patient was sitting in the gallery fire appeared on opposite side. The lights went out air was full of thick smoke. Patient fell with people on top of her. She struggled downstairs semiconscious water sprayed on her. Somebody carried her out.

*Condition on Arrival* Profound shock, respiratory embarrassment with scattered bronchi.

*Extent of Burns* Second degree: Face hands. Total 4%.

*Plasma Therapy* Two units on first day. *Blood Pressure* Systolic 90-90 for brief period, otherwise normal. *Laboratory Data* Maximum hematocrit 40% on first day. Elevated blood chloride 100 mEq. at 37 hours. All other determinations normal.

*Clinical Course* Improved rapidly. *Pulmonary Complication* Atelectasis chest signs. Roentgenogram showed evidence of some trapped air from third to tenth days. *Subsequent Transfusions* None.

*Chemotherapy* Sulfadiazine for first nine days. *Operations* None.

*Discharge* On tenth day burns healed chest clear.





# Index

## A

Administration, problems of	3, 9	14
Adrenal cortical extract, in burn shock		125
Airway foreign matter in removal of		24
inadequate anoxia caused by		28
pressure in		29
resuscitation and sedation of patients with burns which include the		25
Albuminuria in burn shock		170
and resorption of edema		121
Alkalosis in burn shock		112
Anemia, control of		105
Anesthesia, kinds used,		105
Anoxia, in burns		26
caused by inadequate airway		24
Antitetanic serum		5
Arms burns of		108
Asepsis		95
Asthma		36
Atelectasis, areas of		50
Auer, Joseph C. Pulmonary complications a clinical description		34

## B

Back, burns of		109
Bacteriostasis, Chemotherapy in		123
Balance nitrogen		143
potassium		146
Bandage, pressure		90
Bath, melted paraffin wax, in treatment of burns		111
Berchard, Henry K. Resuscitation and sedation of patients with burns which include the air way		25
Bereavement, reactions to		18
Bleeding, in burn shock		122
Blood, nonprotein nitrogen determination of in burn shock		119
Blood bank		128
Blood levels, sulfonamide		94

Blood plasma, administration of		5
Blood transfusions in control of anemia		105
Boric ointment in treatment of surface burns		89
Breathing bronchial absence of		36
Bronchiolitis		35
Bruckley, William J. Pathology with special reference to the pulmonary lesions		65
Bronchospasm, treatment of		20
Bruns, Austin M. Pulmonary complications a clinical description		34
Bunyan envelope use of in third degree burns		104
Burn shock, alkalosis in		123
complicated by pulmonary damage		115
definition of		117
Burns airway resuscitation in		25
sedation in		25
anoxia in		26
chemical in lungs		34
comment on		109
delayed reactions in		31
diet in		105
fear in		25, 28
hyperactivity in, causes of		25
hysteria in		25, 28
pain in		25, 27
physical therapy in		111
comment on		112
exercises in		112
massage in		111
methods of		111
results of		112
teaching relaxation in		112
pulmonary incidence of		34
shock in, complicated by pulmonary damage		115
surface, cleansing of		87
debridement unused in		87
splinting in		90
treatment of		5, 85
administrative advantages of		91
comment on		92
conclusions on		93



# Index

## A

Administration, problems of	39	14
Adrenal cortical extract in burn shock	125	
Airway foreign matter in, removal of	28	
inadequate anoxia caused by	28	
pressure in	29	
resuscitation and sedation of patients with burn which include the	25	
Albuminuria in burn shock and resorption of edema	120	
Alkalosis in burn shock	112	
Anemia control of	10	
Anesthesia, kinds used,	105	
Anoxia, in burns caused by inadequate airway	26	
Antitetanic serum	5	
Arms burns of	108	
Asepsis	95	
Asthma	36	
Atelectasis, areas of	50	
ATK, JOSEPH C. Pulmonary complications a clinical description	34	

## B

Back, burns of	109
Bacteriostasis, Chemotherapy in	123
Balance, nitrogen potassium	143
potassium	146
Bandage, pressure	90
Bath melted paraffin wax in treatment of burns	111
BEZLER, HENRY K. Resuscitation and sedation of patients with burns which include the airway	25
Bereavement, reactions to	18
Bleeding in burn shock	122
Blood, nonprotein-nitrogen determination of, in burn shock	110
Blood bank	128
Blood levels, sulfonamide	94

Blood plasma administration of	5
Blood transfusions in control of anemia	102
Boric ointment in treatment of surface burns	89
Breathing bronchial absence of	26
Bronchitis	35
BUCKLEY, WILLIAM I. Pathology with special reference to the pulmonary lesions	65
Bronchospasm, treatment of	29
BUTTS, AUSTIN M. Pulmonary complications a clinical description	34
Bunyan envelope use of in third degree burns	104
Burn shock, alkalosis in complicated by pulmonary damage	115
definition of	117
Burns, airway resuscitation in sedation in	25
anoxia in	26
chemical in lungs	34
comment on	100
delayed reactions in	31
diet in	105
fever in	25, 28
hyperactivity in, causes of	25
hysteria in	25, 28
pain in	25, 27
physical therapy in	111
comment on	112
exercises in	112
massage in	111
methods of	111
results of	112
teaching relaxation in	112
pulmonary incidence of shock in, complicated by pulmonary damage	115
surface, cleansing of	87
debridement unused in	87
splinting in	90
treatment of	5, 85
administrative advantages of	91
comment on	92
conclusions on	93

# INDEX

medical advantages of	91	Dehydration regimen, in burn shock	119
rationale in	87	Diet, in burn cases	105
results of	92	Dilatation, acute, of stomach	62
with boric ointment	89	Disturbances, mood	21
use of pressure bandage in	90	Donors, blood	130
thermal, in lungs	34	Dyspnea	34
third-degree	103		
distribution of	103	E	
operative procedures in	105	Ears, burns of	109
progress of	108	Edema	34
rehabilitation of patients with	103	pulmonary	60
results from	108	Emphysema	36
separation of slough in	104	areas of	57
splinting of hands in	104	Extremities, burns of	108
treatment of local wound in	103		
		F	
C		FANON, NATHANIEL W Problems	
Calcium, urinary excretion of	146	of hospital administration	3
CANNON, BRADFORD Procedures in		Fear	28
rehabilitation of the severely		Fingers, burns of	108
burned	103	Forehead, burns of	109
CANNON, IDA M Social service ac-		Foreword to symposium on the Co-	
tivities	9	cconut Grove Disaster	1
Canteen, Red Cross, services of	6		
Carbon monoxide poisoning	30	G	
Chemotherapy, and infection, prob-		Gases, complications from inhalation	
lems of	94	of	2
in treatment of burns	123	Gauze, fine-mesh, use of, in third-	
Chronograms, interaction 20, 21,	22	degree burns	103
Churchill, Dr E D,	1	Grafting, skin	106
Civilian Defense, in emergency	3	donor areas for	105
COBB, STANLEY Neuropsychiatric		preparation for	104
observations	14	Grafts, split-thickness	106
Committee, Ladies' Visiting, services			
of	6	H	
War Service, services of	6	Hands, burns of	108
Complications, pulmonary a clinical		splinting of	104
description	34	Helium	30
COPE, OLIVER Management of the		Hemoglobinuria, in burn shock	121
Cocoanut Grove burns at the		Hospital administration	3
Massachusetts General Hos-		Hyperactivity, in burns, therapy of	26
pital, foreword	1	Hysteria	28
COPE, OLIVER Metabolic observa-			
tions	137	I	
COPE, OLIVER Problem of burn shock		Identification, difficulty of, in women	4
complicated by pulmonary		Infarcts	60
damage	115	Infection, and chemotherapy, prob-	
COPE, OLIVER Treatment of the sur-		lems of	94
face burns	85	conclusions on	102
Cyanosis	34	discussion on	101
		problems of	94
D			
Debridement, in surface burns	87		

# INDEX

prophylaxis of	96		
shock in	94		
treatment of	96		
Injury types of	1		
Intratracheal Intubation	28		
Intravenous fluid therapy	9		
Intubation, Intratracheal	28		
K			
1 Ketosteroid, definition of	137		
normal value of	146		
urinary excretion of	146		
Kidney function, maintenance of in			
burn shock	119		
L			
Legs burns of	109		
Lesions parenchymal	66		
pulmonary	41 65		
follow up of	62		
Lesions roentgenologic report of			
pulmonary	41		
LINDEMANN ERICH Neuropsychia-			
tric observations	14		
Lungs, atelectasis in	50		
burns of conclusions of	40		
changes in, chart showing roent-			
genologic course of	101 54 55		
complications of	34		
classification of	37		
damage to complicated by burn			
shock	115		
drowned	58		
edema in	60		
emphysema of	57		
infarcts in	60		
injury to compared to Cleveland			
Clinic Fire of 1929	2		
course and treatment outlined			
	25, 34, 41 65		
lesions of	41		
from chemical burns	34		
discussion on	63		
follow-up of	62		
pathology in	65		
summary of	63		
from thermal burns	34		
miliary mottling in	58		
therapy of difficulty in	39		
LYONS, CHAMP Problems of infec-			
tion and chemotherapy	94		
		M	
		Maey Josiah, Jr., Foundation, con-	
		tribution of	2
		Mallory TRACY B Pathology	
		with special reference to the	
		pulmonary lesions	65
		Management of the Coconut Grove	
		burns at the Massachusetts	
		General Hospital	1
		Massachusetts Women's Defense	
		Corps	6
		Metabolic observations	137
		Metabolism, clinical correlations on	149
		comment on	149
		experiments in	142
		studies in	137
		summary of	155
		Miliary mottling areas of	58
		MOORE FRANCIS D Note on the	
		thrombophlebitis encountered	131
		Mortality	1
		Mottling miliary areas of	58
		N	
		NATHANSON IRA T Metabolic ob-	
		servations	137
		Neck, burns of	109
		Neuropsychiatry	14
		Nitrogen urinary excretion of sex	
		incidence in	146
		Nitrogen balance	143
		figures on, basis for construction	
		of	143
		Nurses Aides services of	6
		Nutrition importance of in burn	
		cases	110
		maintenance of in burn shock	124
		O	
		Office of Scientific Research and De-	
		velopment, U S Govern-	
		ment	1
		Ointment, boric	89
		Ointment, boric-acid, use of in third	
		degree burns	103
		scarlet red	105
		Oxygen, anoxia, from inadequate	
		transport of by blood	30
		Inspiration of	29

# INDEX

Oxygen tent	34	Pulmonary lesions, with special reference to the, Pathology	65
Oxygen therapy	5	roentgenologic report of the	41
P			
Pain	27	R	
control of, in burn shock	115	Radiograms, mobile condenser discharge unit in, use of	41
Pathology with special reference to the pulmonary lesions	65	Râles, increase of	34
Penicillin, temperature response following use of	95	Red Cross, services of	6
therapy with	98	Red Cross Disaster Relief	12
Phlebothrombosis, definition of	132	Rehabilitation of the severely burned, procedures in	103
Phosphorus, urinary excretion of	146	Restlessness	34
Physical therapy, in burns	111	Resuscitation and sedation of patients with burns which include the airway	25
PITTMAN, HELEN The pulmonary-complications a clinical description	34	RHINELANDER, FRÉDÉRIC W Problem of burn shock complicated by pulmonary damage	115
Plasma, dried	128	Roentgenologic report of the pulmonary lesions	41
frozen	128	analysis of	50
remaking of	130	ROURKE, G MARGARET Metabolic observations	137
supply of	129	S	
handling of	130	Scalp, burns of	109
system of administration of	129	SCHATZKI, RICHARD Roentgenologic report of the pulmonary lesions	41
system of filtering	130	Scientific Research and Development, Office of, U S Government	1
veins used for administration of	129	Sedation, morphine	95
Plasma therapy, in burn shock	116	and resuscitation of patients with burns which include the airway	25
Poisoning, carbon monoxide	30, 65	Serum, antitetanic	5
Postmortem studies	65	17-Ketosteroid, definition of	137
discussion of	84	normal value of	146
Postmortem studies, summary of	80	urinary excretion of	146
Potassium balance	146	Shock	30
Precautions, sterile	95	burn, adrenal cortical extract in	125
Pressure, in airway	29	albuminuria in	120
Pressure bandage	90	bleeding in	122
Problem, of burn shock complicated by pulmonary damage	915	complicated by pulmonary damage	115
of hospital administration	3	age	127
of infection and chemotherapy	94	conclusions on	115
Procedures in rehabilitation of the severely burned	103	control of pain in	117
Prophylaxis, tetanus	98	definition of	110
Protocols of Coconut Grove disaster patients admitted to the Massachusetts General Hospital	159-165	dehydration regimen in	121
Psychiatry, need for	13	hemoglobinuria in	122
problems of	3, 9, 14	intestinal ulceration in	
Psychoneuroses	17		
Psychotic conditions, psychogenic	16		
Pulmonary complications clinical description	34		
Pulmonary damage in connection with burn shock	115		

# INDEX

Kidney function in, maintenance of	119	Thrombophlebitis	131
manipulation in	116	case reports on	132 136
nonprotein nitrogen determina- tion of	119	conclusions on	136
nutrition and	114	definition of	132
plasma therapy in	116	discussion of	136
pulmonary damage in	115	Toxins	85
Slough, separation of	104	Tracheotomy	29
Social Service	9	Treatment, of casualties	3
SOUTTER, LAMAR A note on the blood bank	128	principles of	94
Splinting	90	of surface burns	85
Stomach, acute dilatation of	61		
Sulfadiazine in treatment	5	U	
Sulfadiazine levels, determinations of	97	Ulceration, intestinal in burn shock	122
Sulfonamide blood levels	94	U S Government Office of Scientific Research and Development	1
Sulfonamide therapy	96	Urine boric acid excretion in	90
complications arising from	95	calcium excretion in	146
Surface burns, treatment of	85	nitrogen excretion in	143
		phosphorus excretion in	146
		17-ketosteroid excretion in	146
T		W	
Tetanus prophylaxis	98	WATKINS ARTHUR L. A Note on physical therapy	111
Therapy oxygen	5	WILSON HILDEGARD Metabolic ob- servations	137
physical, in burns	111	Women identification of difficulty in	4
plasma, in burn shock	116		
sulfonamide	96		
Thermotherapy	111		





